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PHYSIOLOGY AND ETIOLOGY

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WASHINGTON, D. C.

IT IS innate in most humans to want to see clearly, not only with one eye but with both eyes simultaneously. Clear vision is possible when the object receiving the visual regard is imaged sharply on the fovea. If the object is near to the eye or the eye is hypermetropic, accommodation is required to make the image sharp. Consequently, clear vision for a very large part is dependent upon accommodation. Clear vision with both eyes can only be attained by having the sharp image projected simultaneously onto each fovea. This is known as bifoveal fixation. If the object is near the eyes, convergence is necessary in order for its image to be projected onto each fovea. Therefore, bifoveal fixation, to a great degree, relies upon convergence. To be more specific, it can be stated that clear bifoveal vision for an object near the eyes demands that accommodation and convergence occur simultaneously.

ASSOCIATION OF ACCOMMODATION AND CONVERGENCE

Perhaps this is the reason accommodation and convergence are associated in the sense that a certain amount of accommodation reflexly calls forth a precise quantity of convergence, and vice versa. This association will be referred to as the accommodation-convergence association reflex. The detailed nature of the accommodation-convergence association reflex varies from individual to individual with each possessing a specific pattern which, as far as I can determine, appears to be relatively unchangeable. This association reflex brings forth accommodation and convergence in a constant ratio. The specific ratio by which accommodation and convergence are reflexly associated can be determined for each individual, being most conveniently measured in the following units: diopters for accommodation and meter angles for convergence. The commonest ratio is in the neighborhood of one to one; one diopter of accommodation being reflexly associated with approximately one meter angle of convergence. The ratio of 1:1 is ideal for an emmetrope who is orthophoric, because the proper amount of accommodation required for clear vision is reflexly associated with the correct degree of convergence needed for bifoveal fixation, regardless of the location of the object receiving the visual attention.

DISSOCIATION OF ACCOMMODATION AND CONVERGENCE

Seldom does the ideal reflex association of accommodation and convergence exist which provides simultaneously clear vision and bifoveal fixation. If the accommodation provides clear vision, the degree of convergence reflexly associated with it usually will not facilitate bifoveal fixation. Or if the convergence is that required for bifoveal fixation, the accommodation reflexly associated with it usually will not permit clear vision. Therefore, theoretically an adjustment of either the accommodation or the convergence is demanded in practically all individuals prior to attaining clear bifoveal vision. Such an adjustment is referred to as dissociation of accommodation and convergence.

Dissociation is performed through the medium of the fusional vergence reflex in

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the following manner. The average individual locks the accommodation at the required amount to obtain clear vision, and this is reflexly associated with a certain ratio of convergence. Most likely, the degree of convergence associated with the fixed amount of accommodation will not afford bifoveal fixation. The convergence is then sufficiently altered by the vergence reflex to provide bifoveal fixation, the clear vision persisting since the accommodation remains unchanged. Therefore, the accommodation-convergence association reflex and the fusional vergence reflex, in most individuals, work hand-in-hand in order to supply clear bifoveal vision.

PATHOPHYSIOLOGY OF ACCOMMODATIVE ESOTROPIA

Accommodative esotropia develops when the amount of accommodation required for clear vision is associated reflexly with such an excessive degree of convergence that the fusional vergence reflex is unable to overcome it in order to establish bifoveal fixation. The train of events producing accommodative esotropia is important. First, there is the desire by the patient to acquire clear vision. Second, in order to secure clear vision, some particular visual circumstance is present that requires accommodation. Third, the patient lacks the amount of accommodation required for clear vision and through the medium of the accommodation-convergence association reflex an excess of convergence issues forth over and above that amount needed for bifoveal fixation. Fourth, the patient has the desire for bifoveal fixation as well as the urge for clear vision. Fifth, fusional divergence is applied by the fusional vergence reflex; however, it is unable to overcome the magnitude of excessive convergence. Sixth, at this point the patient is forced to choose between clear vision or bifoveal fixation. If clear vision is the choice, accommodative strabismus prevails. If bifoveal fixation is the choice, the excessive convergence must be reduced to a point, at least, that is capable of being overcome by fusional divergence. The only way in which the excessive convergence can be reduced is by diminishing the amount of accommodation that was required for clear vision. Consequently, through the medium of the accommodation-convergence association reflex the associated excessive convergence is lessened. This in turn blurs the vision; however, the choice of bifoveal fixation is now realized.

ETIOLOGY OF ACCOMMODATIVE ESOTROPIA

Accommodative esotropia is due to either of two principal causes. First, the accommodation-convergence association reflex calls forth an excessive amount of convergence over and above that required for bifoveal fixation while accommodating to see clearly. Second, the fusional vergence reflex is not supplying sufficient fusional divergence to reduce the excessive convergence to the point that bifoveal fixation is possible.

The accommodation-convergence association reflex may be normal or abnormal while causing accommodative esotropia. A normal accommodation-convergence association reflex causes accommodative esotropia in a hypermetrope. Accommodation sufficient to provide clear vision in the hypermetrope will have an excess of convergence over that amount needed for bifoveal fixation associated with it. If the excess convergence is not diminished by the fusional divergence so that bifoveal fixation is possible, accommodative esotropia is the consequence.

An abnormal accommodation-convergence association reflex of a certain type causes accommodative esotropia in myopes, emmetropes, or hypermetropes. What is a normal? What is an abnormal? And of what type must the abnormal accommodation-convergence association reflex be in order to cause accommodative esotropia? A normal association of accommodation and convergence consists of approximately the ratio of 1 : 1, the accommodation measured in diopters and the convergence in meter

angles. A limited departure from the ratio of 1 : 1 in either direction is considered within normal limits. However, there is definitely a departure point from the 1 : 1 ratio that is considered abnormal. An example of an abnormal accommodation-convergence ratio is 1 : 3, that is, 1 diopter of accommodation associated with 3 meter angles of convergence. A second example is just the reverse, that is, 3 : 1, or 3 diopters of accommodation associated with 1 meter angle of convergence. If the abnormal accommodation-convergence ratio is of the type that a greater number of meter angles of convergence are associated with a lesser number of diopters of accommodation, such as a 1 : 3 ratio, the situation is ripe for accommodative esotropia. Any accommodation whatsoever occurring in a patient afflicted with this type of abnormal accommodation convergence reflex will provoke an excess of convergence. Insufficient fusional divergence to overcome the excessive convergence precludes bifoveal fixation, thus causing accommodative esotropia.

The more accommodation required for clear vision, the larger the excess convergence will be that results from such an abnormal reflex. Since the near objects require more accommodation in order to see them clearly, the accommodative esotropia caused by such an abnormal reflex will more often occur only while looking at a near object. If the accommodative esotropia is present while looking at a distance, as does happen in a hypermetrope, the esotropia will be much greater while looking at near in the presence of this type of abnormal accommodation-convergence association reflex.

The manner in which this abnormal reflex is identified is as follows. The findings of prism and cover measurements for distant and near fixation are compared. Certain conditions must be controlled while doing the prism and cover measurements, but principally, the accommodation must be controlled. This is accomplished by doing two things. First, the refractive error is neutralized fully with lenses. Second, the patient fixates and keeps clear a small symbol; for instance, of a size equivalent to a 20/30 Snellen letter at six meters, and to a 20/30 Lebensohn letter at one-third meter. To make sure that the symbol is kept clear, it is best to present new symbols repeatedly, demanding the patient to identify each new symbol presented.

A normal accommodation-convergence reflex association exists when the prism and cover measurements are approximately equal for distant and near fixation. An abnormal reflex of the type that causes accommodative esotropia will reveal a much greater convergence by prism and cover determinations at near than at distance. A significant abnormal reflex will be recognized when measured in accord with the above detailed instructions, by there being no esotropia while fixating a distant letter, but esotropia being manifest upon fixating a near letter.

THE FUSIONAL VERGENCE REFLEX

In addition to the accommodation-convergence association reflex causing accommodative esotropia, the fusional vergence reflex, in not fulfilling the function for which it was designed, may be implicated in the development of accommodative esotropia. The fusional divergence portion of the fusional reflex may be entirely adequate to overcome any excessive convergence that might develop with the application of the accommodation-convergence reflex, yet esotropia may occur in either of the two following conditions. First, the patient may on occasions be unwilling to use the available fusional divergence because of the effort involved in obtaining bifoveal fixation. Particularly is this so when the patient is fatigued, ill, or just awakening from a deep sleep. At such times the eyes are seen to "cross," yet while the child is well rested and physically fit, the eyes are straight. Also heterophorias, in addition to the esophoria reflexly associated with accommodation, impose an additional load on the already

overtaxed vergence reflex. Heterophorias, then, are another factor which contributes toward making the patient unwilling to put forth the effort required to effect the corrective movement needed for bifoveal fixation.

The second condition that precludes the application of an available adequate fusional divergence is a barrier to equal foveal fixation. The drive for bifoveal fixation is probably due to the desire to have each fovea simultaneously supply the visual perception centers with identical information regarding the clarity of the image of the object of regard. This desire, then, represents the stimulus for the fusional vergence required to make bifoveal fixation possible. Any ophthalmologic condition which precludes equal foveal vision would consequently lessen the desire for bifoveal fixation. Obstacles that prevent equal foveal vision are anisometropia and unilateral corneal haze, cataract, vitreous haze, muscular lesion and affliction of the papillomacular bundle.

The fusional divergence portion of the fusional reflex may be grossly inadequate to cope with the excessive convergence issued forth by the accommodation convergence association reflex. The inadequacy of the fusional divergence may be absolute, that is, the fusional divergence is completely lacking, Or the fusional divergence amplitude may be smaller than normal and consequently be unable to overcome a relatively small amount of excessive convergence, which a more adequate fusional divergence would have no trouble coping with.

The fusional divergence amplitude which attempts to overcome the excessive convergence that results from accommodating to see clearly is not the well-known divergence amplitude that is determined with prism while fixating a light. In such a determination the degree of accommodation is ignored. The examiner is merely measuring the amount the eyes can diverge irrespective of the state of accommodation. The fusional divergence amplitude which overcomes the excessive convergence resulting from the accommodation-convergence association reflex is that amount of divergence available while the degree of accommodation remains at a constant figure. This quantity of fusional divergence is determined relative to a specific amount of accommodation. Consequently, I shall call this the relative fusional divergence amplitude.

It is possible to directly determine the relative fusional divergence amplitude with either prisms or a haploscopic device. Since the average ophthalmologist does not possess, or is not facile at operating, a haploscopic instrument, prisms must be depended upon while directly determining the relative fusional divergence amplitude. It is so extremely difficult for me to control the patient's accommodation when using prisms while attempting this determination that I do not feel my findings are sufficiently reliable to justify the attempt. Furthermore, the important information the ophthalmologist desires in treating accommodative esotropia is the diopters lens power required to provide clear bifoveal vision. Since prism determination does not afford this necessary information, I see no reason for the ophthalmologist to become interested in the direct measurement of the relative fusional divergence amplitude.

However, a system will be described in which the amplitude of relative fusional divergence is indirectly measured by using diopter lens power; a system which yields the fruitful clinical information of the particular diopter lens power required to assist the accommodative esotrope to experience clear bifoveal vision. This indirect system of measuring the amplitude of relative fusional divergence is possible because of the following principles. Due to the accommodation-convergence association reflex, either increasing or decreasing the accommodation, while maintaining a constant meter angle of convergence, will induce a

different associated phoria with each variance of the accommodation. So long as the induced phoria is capable of being overcome by the fusional vergence reflex, the constant meter angle of convergence can be maintained. The increasing esophoria that develops when the accommodation is increased while the constant meter angle of convergence of the eyes is maintained must be countered by the relative fusional divergence amplitude. Esophoria no longer is existent when the induced convergence exceeds the amplitude of the relative fusional divergence; accommodative esotropia has developed. Decreasing the accommodation the least bit will in turn reduce the associated convergence to the point at which it barely fits within the amplitude of the relative fusional divergence. Now esophoria replaces esotropia. A prism and cover determination at this point, while the patient is accommodating the controlled amount, will be a rough determination of the relative fusional divergence amplitude.

The two important details for indirectly determining the amplitude of relative fusional divergence are controlling the accommodation as it is varied by designated amounts and controlling the degree of convergence as it remains constant. These details are accomplished by the following steps:

1. The patient's refractive error is completely neutralized with lenses.

2. A fixed degree of convergence of the patient's eyes is selected and enforced by the examiner. I particularly like to select 3 meter angles of convergence for the determination. This degree of convergence is enforced by requiring the patient to fixate bifoveally a symbol which is equivalent in size to a 20/30 Lebensohn letter. The examiner is certain that the patient is bifoveally fixating the symbol if there is no movement of the eyes while doing the cover-uncover test.

3. The accommodation is controlled by demanding the patient to identify the small symbol presented at one-third meter. In order that the accommodation continue to be stimulated a controlled amount, new symbols should be repeatedly presented to the patient.

4. Trial case lenses of equal power are superimposed before each eye over the refractive error correction. The accommodation must be varied proportional to the power of the lenses superimposed if the fixation symbols are to be identified.

a. If clear vision and bifoveal fixation are present at both 6 meters and one-third meter prior to superimposing the lenses, minus lenses are superimposed. This requires an increase in the accommodation in order to identify the symbol. The increased accommodation is associated with an urge to increase the degree of convergence because of the accommodation-convergence association reflex. The urge to increase the convergence becomes an esophoria, since it is negated by the relative fusional divergence as long as the convergence is kept constant. Minus lenses of increasing strength are superimposed until the urge to increase the convergence exceeds the relative fusional divergence amplitude, at which point accommodative esotropia will be present if the symbol is identified.

b. If clear vision and bifoveal fixation are present at 6 meters, but clear vision and esotropia present at one-third meter, plus lenses are superimposed before the refractive error correction. This reduces the accommodation required to identify the small symbol, thereby diminishing the convergence. At the point where sufficient plus lens power has been superimposed to reduce the convergence so that it fits within the amplitude of relative fusional divergence, accommodative esotropia will be replaced by esophoria since the symbol becomes bifoveally fixated.

5. A prism and alternate cover determination of the esophoria present while accommodating through the maximal minus

or the minimal plus superimposed lenses that permit clear bifoveal vision, as the case may be, is an indirect rough measurement of the amplitude of relative fusional divergence.

It is of importance to the ophthalmologist to determine indirectly the relative fusional divergence amplitude by using diopter lens power while clinically dealing with accommodative esotropia. Since there are more causes of accommodative esotropia than simple hypermetropia, there is more involved in the spectacle treatment of accommodative esotropia than just neutralizing the hypermetropia with lenses. For example, accommodative esotropia resulting from an abnormal accommodation-convergence association reflex is also amenable to being controlled with spectacles in the same way that accommodative esotropia due to hypermetropia is controlled. In such an example, solely neutralizing any attendant hypermetropia with lenses does not control the accommodative esotropia. This problem can only be curbed by those which are bifocals, if depending on spectacles. The ideal power of the near "add" portion of the bifocal lenses is the minimum that will permit clear bifoveal vision at one-third meter. This ideal power of the near "add" is determined by indirectly measuring the amplitude of relative fusional divergence and at the same time noting the power of the minimum plus lenses, which were superimposed before the patient's ametropic correction, that permitted clear vision and bifoveal fixation. In other words, the difference between the maximum amount this type patient can accommodate while retaining bifoveal fixation at one-third meter and the 3 diopters of accommodation required for clear vision at one-third meter represents what the minimum power of the near "add" should be. For example, an emmetrope has an abnormal accommodation-convergence association reflex of the type in which an excessive amount of convergence is associated with each unit of accommodation. The vision is perfect and orthophoria is present while fixating at 6 meters. While seeing clearly at one-third meter, esotropia of 30 prism diopters develops. Plus one diopter lenses placed before the eyes reduce the esotropia to 20 prism diopters while the 20/30 Lebensohn symbol is identified at one-third meter. Plus one and one half diopters lenses reduce to 15 prism diopters the excessive convergence associated with the one and one half diopters of accommodation required to see clearly at one-third meter. The 15 prism diopters of excessive convergence is within the amplitude of relative fusional divergence which neutralizes it from an esotropia to an esophoria. Thus, plus one and one half diopter lenses in this example permit clear bifoveal vision at one-third meter and consequently are the minimum plus power which the near "adds" ideally should be.

The orthoptic technician is more blessed than the average ophthalmologist since the amplitude of relative fusional divergence can be directly determined with ease on a haploscope. In such an instrument the accommodation can be controlled rather accurately while reading foveal target material on slides, and at the same time the fusional divergence is measured by moving the arms of the machine. Acquiring a direct measurement of the amplitude of relative fusional divergence is of great value to the orthoptist since an abnormal measurement requiring treatment would be revealed as well as a standard established by which progress of treatment could be measured.

An accommodative esotrope with bifoveal fixation ability can be attacked by orthoptics, in one of two directions. One, the accommodative esotrope can be taught to substitute bifoveal fixation in place of clear vision. This is substitution therapy since one accomplishment is gained at the expense of sacrificing another. There is a legitimate place for such substitution therapy. Two, orthoptics can increase the rela-

tive fusional divergence amplitude so that clear vision and bifoveal fixation become simultaneously possible. So far as I can determine, no treatment, orthoptics included, alters the manner in which accommodation and convergence are reflexly associated. If orthoptics improves accommodative esotropia, the improvement can be measured by recording an increase in the relative fusional divergence amplitude.

Symposium: Accommodative Esotropia

CLINICAL COURSE AND MANAGEMENT

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THE RECOGNITION, evaluation and management of accommodative esotropia is difficult, since its manifestations are varied and the professional attitude toward the condition differs greatly from one geographical location to another, and among individuals within the same geographical location. This confusion is not simplified by the fact that there is not a universally accepted definition of accommodative esotropia or of other relevant terminology.

When examining cases of esotropia, three distinct subgroups will be recognized. First, there is the constant deviation, which has been variously called "mechanical," "fixed," or "nonaccommodative." This patient has a constant deviation not materially influenced by the full correction of the refractive error. Second, there is the esotrope who, under some circumstances, has straight eyes, and under others, has a variable deviation. This is the so-called accommodative or intermittent esotrope. Third, there is a quite large group of persons who have some element of each, both accommodative and nonaccommodative. A certain irreducible minimum of deviation is present (the nonaccommodative element) and superimposed upon this is a variable element, which seems to vary with the amount of accommodation being used and circumstances of its use (the accommodative element). Our discussion will be limited to the intermittent or accommodative group.

As has been recognized by Dr. Parks' discussion, accommodative esotropia may be divided into two chief groups. They are (1) those associated with an excessive hypermetropia and having a normal accommodation-convergence ratio, and (2) those usually associated with a smaller hypermetropia, but having an abnormal accommodation-convergence ratio. It should be recognized that the size of the refractive error, the accommodation-convergence ratio, the adequacy of the relative fusional divergence, the state of the foveal vision, and the size of the basic heterophoria may cause certain intermediate or atypical clinical patterns. The presence or absence of bifoveal fixation is of especial significance in management and prognosis. Nonetheless, most cases of accommodative esotropia are due either to the hypermetropia present or to an abnormal accommodation-convergence relationship which is inadequately controlled, and they will be so discussed.

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HISTORY AND COURSE

The typical history of accommodative esotropia is that of an intermittent deviation which was usually first noticed between the ages of 18 months and 5 years. The occurrence may be infrequent at first, usually at times of fatigue, emotional upset, illness, or with near activity. The onset is, at times, accompanied by a period of visual confusion, actual diplopia, awkwardness, or irritability, which, however, usually lasts only from a few days to a few weeks.

As time goes on, this intermittent deviation may follow one of several courses. First, there may be a continuing intermittency for both distant and near gaze, being present only when the patient is accommodating, and alleviated when accommodation relaxes. Second, it is possible that the patient may continue to have

straight eyes for distant gaze but be almost constantly esotropic at near gaze, particularly when accommodating. Third, the intermittent deviation may become increasingly frequent until after a time, the deviation is constantly present. A portion of these last may be relieved by the full hypermetropic correction, at times associated with orthoptics; while in others the constant deviation will persist in spite of wearing a full hypermetropic correction. It is apparent that this last subgroup has developed a nonaccommodative element, though initially it seemed completely accommodative in nature.

The reasons for the difference in behavior in various early intermittent esotropes are not fully apparent. However, the basic esophoria, the size of the refractive error and the state of the relationship between accommodation and convergence would seem to be major factors in their behavior, as has been emphasized by Dr. Parks.

CLINICAL FINDINGS

1. *Accommodative Esotropia Having a Normal Accommodative-Convergence Ratio*

I would like to point out, as I go along, that in the two major groups there are certain very definite points of similarity but there are also two or three outstanding points of dissimilarity, and the points of dissimilarity are important because they influence so profoundly the approach and the treatment of this problem.

The deviation may behave in one of two ways in the group having a normal accommodative-convergence ratio. The patient may have straight eyes for distance and near until attempting to see clearly, at which time the esotropia occurs. However, binocular alignment is restored as soon as the accommodation relaxes. On the other hand, certain cases may initially follow such an intermittent pattern but soon become constantly esotropic when not wearing glasses, even when not accommodating. However, the deviation will be eliminated by the proper refractive correction. The characteristic of the entire group is that the proper single hypermetropic correction will alleviate the esotropia.

The deviation in this group occurs in an individual who has a significant hypermetropia (2 to 6 diopters) often associated with astigmatism. Since the accommodation-convergence relationship is normal, the deviation is relieved by a full hypermetropic correction and the eyes are straight for both distant and near gaze.

In the accommodative esotropia with normal accommodation-convergence ratio, the absolute accommodation and the accommodation-convergence relationship are usually normal. The problem is not one of a deficient accommodation mechanism, nor of one poorly balanced with convergence. Rather it is one of a normal mechanism being excessively stimulated through a large hypermetropic error, and responding with an undesired convergence.

Since the onset of the deviation is usually after the age of 2 years, amblyopia is not severe and responds well to treatment unless a significant anisometropia or organic reduction in vision is present. The presence of anisometropia jeopardizes a final good visual result, somewhat in proportion to the degree of anisometropia present. For example, a higher percentage of patients having a refractive difference of 2 diopters will be amblyopic than those having a difference of only 1 diopter in the two eyes. The age of onset of the strabismus, the duration of the deviation, and the amount of anisometropia would seem to be significant factors in the cause and the treatment of amblyopia.

Retinal correspondence is normal, since the onset of the deviation is relatively late and binocular single vision has had some opportunity for early reinforcement. However, the persistence of a variable deviation does make suppression a frequent finding, especially foveal suppression, and the overcoming of this defect is a major item in treatment.

In general, fusion and fair fusional vergences can either be demonstrated early during the period of observation or may be easily restored and enhanced in this form of strabismus.

The clinical picture of the average accommodative esotrope with normal accommodation-convergence ratio is, then, that of a child who has a variable esotropia beginning after the age of 2 years, which is compensated for distance and near by a moderate to high hypermetropic correction. Amblyopia is not severe. Suppression is present but can be overcome. Fusion is present or can be easily restored. The prognosis is good.

II. *Accommodative Esotropia with Abnormal Accommodation-Convergence Ratio*

The behavior of the deviation is quite characteristic of this type of strabismus. The eyes are straight for distance, though at times a small intermittent esotropia may manifest itself, especially when the patient's refractive error is not fully corrected and he is visually attentive to details. However, when attempting to see *any* visual detail at 13 inches, there is a large esotropia, even when the full distance correction is being worn. This deviation, however, may be alleviated by the addition of a plus sphere to each eye. Now, having clear vision with a minimum of accommodation, the eyes are straight for distance and near, and the diagnosis is confirmed.

When examined under atropine, the so-called abnormal accommodation-convergence ratio case demonstrates an insignificant refractive error, often less than 1 diopter of either hypermetropia or myopia.

Since the refractive error is not abnormal in size, other causes for the deviation must be found. The accommodation, when tested, may show several things of interest. *First,* the accommodation-convergence ratio is abnormal, i.e., a given amount of accommodation calls forth an excessive amount of convergence. *Second,* the amplitude of relative fusional divergence is insufficient to compensate for the convergence present. *Third,* the near point of accommodation or punctum proximum of accommodation (presumably a measure of the absolute accommodation) is, in my experience, frequently remote. This suggests some basic defect in the accommodation mechanism.

Whether the convergence in these cases is due to an exaggerated response to normal accommodative effort, or whether it is due to an excessive stimulation of a weakened accommodation, cannot at this time be definitely stated. Possibly both are factors in the accommodative esotropia with abnormal accommodation-convergence ratio.

Similar to the normal accommodation-convergence ratio type, since onset is after the age of 18 months, amblyopia is not severe, and visual acuity is rather readily restored. Suppression is present but not deep-seated because of the variability of the deviation.

Fusion is potentially good, but the fusional vergences, especially when measured relative to a given amount of accommodation, are inadequate for the amount of convergence called forth. This presents a major problem in treatment.

The outstanding characteristics of this group are, then, (1) an insignificant refractive error, (2) small or no deviation at 20 feet, and a larger variable deviation at 13 inches, (3) an abnormal accommodation-convergence ratio, and (4) inadequate relative fusional divergence for the case under consideration.

It should not be inferred from the description of the two types of esotropia above that all findings are so absolute. Many cases are intermediate between the two types. Thus they would have a moderate hypermetropia and, in addition, while the eyes are straight with full correction at 20 feet, definite esotropia persists at 13 inches until plus spheres are added. This intermediate group has a deviation which is due both to the accommodation induced by the refractive error and to an abnormal accommodation-convergence ratio.

MANAGEMENT

The goals in the treatment of all strabismus are (1) good vision in each eye, (2) good ocular alignment, and (3) good binocular vision under all possible circumstances. As has been pointed out before, deviation of very early onset, delay in treatment, and asymmetry in ocular findings greatly interfere with reaching these goals. More specifically, the presence of anisometropia, incomitance, a nonaccommodative element, or fixation disparity makes treatment more difficult.

Since the case of purely accommodative esotropia has the ability to assume the straight position when circumstances are favorable, the treatment is primarily directed at improving visual acuity, providing the proper spectacle correction to keep alignment secure, improving fusion, and normalizing or making more flexible the relationship of accommodation and convergence.

The treatment of amblyopia will be discussed by Miss Tibbs. Suffice it to say that occlusion should be complete at first, and sufficient to maintain good vision after it has been attained. Amblyopia will recur if anisometropia is present and if fairly secure vision has not been established.

The proper strength spectacles will usually straighten the eyes of all accommodative esotropes. In the type with a normal accommodation-convergence ratio, single convex lenses of the proper strength will be satisfactory. In the abnormal ratio type, bifocals are necessary to allow the proper alignment as much of the time as possible. The earlier and the more constantly the visual axes are aligned, the greater will be the opportunity for establishing secure binocular vision.

The proper strength spectacle is determined as follows: (1) an atropine refraction establishes the amount of the refractive error present; (2) in patients below the age of 4, and at times below the age of 6 years, the full strength must be prescribed since an estimate of the minimal needs cannot be obtained. (3) In children sufficiently mature, a postcycloplegic examination is done. The full cycloplegic finding is placed in a trial frame. Then while the patient is maintaining clear 20/30 vision on the Snellen chart at 20 feet, the hypermetropic correction is simultaneously reduced 0.5 diopter in each eye. Each time a reduction is made, cover-uncover is done to determine whether bifoveal fixation is being maintained. This progressive reduction in sphere is continued until an obvious esotropia is manifest or demonstrated by cover-uncover (not by rapid alternate cover).

The weakest correction which would allow bifoveal fixation and 20/30 vision is considered the correct one.

(4) A similar examination is done at 13 inches, beginning with full cycloplegic correction and a +3.00S add O.U. The progressive reduction is made while the patient is identifying a 20/30 symbol on a near vision chart. Again, the weakest spherical correction allowing bifoveal fixation and 20/30 vision is the proper correction for near. If the minimal needs for 20 feet and 13 inches differ by more than 1 diopter, bifocals should be ordered, incorporating the two minimal corrections. (5) At least once in six months and at times oftener, the "minimal correction" should be determined and the glasses reduced if the change is appreciable. Care should be taken to maintain an adequate strength of lens during spells of prolonged illness and during the winter when the eyes are being used heavily.

(6) The progressive reduction in glasses may be aided by the use of minus fitovers in grades of 0.5 diopter. These are used in conjunction with orthoptic procedure as will be described by Miss Tibbs and Mrs. Kramer. (7) Eventually many accommodative esotropes will maintain bifoveal fixation securely with no refractive help for 20 feet and a reduced strength for 13 inches. At such times reading glasses only are ordered. There is a reasonable hope that any young accommodative esotrope having a hypermetropic error of less than 4 diopters

may eventually be able to discard glasses, at least for general wear.

It should not be assumed that the simple progressive reduction in glasses is adequate treatment. Other things are important. Orthoptics is most important in treating the accommodative esotrope. As will be demonstrated by Miss Tibbs and Mrs. Kramer, the reasonable sequence of orthoptic treatment is as follows: (1) The best possible visual acuity in each eye is obtained (this may be done by the ophthalmologist or the technician under his supervision), (2) suppression is overcome, (3) diplopia is encouraged, (4) fusion is stimulated, (5) fusional amplitudes are improved, and (6) accommodation and convergence are dissociated. This treatment is frequently carried on during the period of time that the progressive reduction of glasses is being carried out.

In general, there are three or four qualifications for the beginning of orthoptic treatment. First, the patient must be sufficiently mature to understand and continue with binocular training over the necessary length of time. Usually this requires that the child be of school age. Second, vision must be sufficiently good in each eye so that binocular training can be reasonably engaged in. This usually requires a vision of 20/50 or better in each eye, though at times certain things may be done when the vision is poorer. Third, the parents must be sufficiently intelligent and interested to offer full cooperation for the home training, as well as getting the child to the office, when necessary. Fourth, and finally, the referring ophthalmologist must have a reasonable understanding of what is being done, and an intelligent interest in furthering the aims of the treatment.

Medication seems to be a reasonable adjunct to some of this treatment. We do not use atropine for treatment. It is our opinion that prolonged atropinization does not prevent accommodative effort. Rather, the constantly blurred image encourages accommodative effort, and with this increased effort, convergence is also exaggerated. Some of us, however, use miotics. Most recently we have been using DFP (di-isopropyl fluorophosphate) 0.02 per cent to 0.03 per cent, one drop daily in each eye. This causes a smaller pupil and some increased ciliary tonus, thus allowing a given amount of accommodation with less accommodative effort. The early results of the use of DFP are encouraging, especially in cases having an abnormal accommodation-convergence ratio with inadequate relative fusional divergence.

While surgery seldom is wise in accommodative esotropia, at times it may be helpful. This is only true if the basic esophoria is at least 10 prism diopters, and the relative fusional divergence is inadequate. Surgery seldom is wise.

It is important to recognize the role played by the general health and the state of the nervous system. A vacation with a minimum of eye activity, a regular daily schedule, and a minimum of stimulating activities such as movies and television, all seem to help the accommodative squinters.

In brief summary, it should be pointed out that there are two chief forms of accommodative esotropia. The normal accommodation-convergence ratio type has a moderate to high hypermetropia which causes an esotropia for distance and near on accommodation and is compensated by the proper single lens. The abnormal accommodation-convergence ratio type has a negligible refractive error, little or no esotropia at 20 feet, but definite deviation at 13 inches on accommodation. This deviation is alleviated by the proper bifocal spectacles. The treatment of accommodative esotropia is the wearing of glasses from as early in life as possible in order to maintain binocular alignment. Amblyopia must be overcome and vision reinforced by periodic occlusion. At the school age, orthoptic procedures will improve fusion and fusional amplitudes, making it possible to reduce progressively the strength of the glasses worn. Thus the goal of good vision and good alignment with the minimum of artificial aid may be frequently attained.

Symposium: Accommodative Esotropia

OFFICE TRAINING

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THE PANEL appreciates the fact that some of the following procedures and technics will not lend themselves to every ophthalmologist's office, but a sufficient number can be done by any office to make their description worth while.

The technician must have an adequate knowledge of the ophthalmologist's findings and an understanding of the orthoptic steps to be followed. A good relationship between the technician, the child and the parent is also of utmost importance.

The goal of orthoptic treatment is (1) securing the best possible vision in each eye, and (2) attaining good single binocular vision by (a) dissociating the function of the accommodation and convergence relationship and (b) establishing the proper harmonious relationship between the two.

The essential findings from the ophthalmologist are (1) the refractive error determined under full cycloplegic, and (2) the prism and cover measurements with correction under cycloplegic while not accommodating. Other helpful findings are: vision O.D. and O.S.; punctum proximum, or the point conjugate to the retina when the eye is accommodating to its maximum ability; amplitude of relative fusional divergence; deviation cc at 20 feet, and sc while accommodating at 20 feet, cc at 13 inches, and sc while accommodating at 13 inches, cc add +3.00S at 13 inches; ocular rotations.

The orthoptic technician should have a working knowledge of refraction. Whether the case is purely accommodative or is accommodative combined with a nonaccommodative element, the orthoptic treatment is the same for the purely accommodative factor.

The reasonable sequence of treatment is as follows:

1. The treatment of amblyopia
2. The overcoming of suppression
3. The stimulating of diplopia
4. The improving of fusional vergence amplitudes
5. Dissociation of accommodation and convergence (relative fusional vergence)

1. AMBLYOPIA

While the amblyopia is not usually of severe degree, the vision must be brought to the best possible level in each eye. The responsibility for this task may fall to the ophthalmologist, or, at times, may be assigned to the orthoptic technician. In general, it is customary to use adhesive occlusion on the face over the fixating eye constantly until the vision has improved to 20/50 or better in the deviating eye. At this point, certain types of occlusion upon the glasses may be used. There are celluloid clip-on occluders of various styles, the multi-facet lens as exemplified by the American Optical Company occluder lens, adhesive tape pasted on the posterior face of the lens, or translucent paper such as Permafilm or music mending tape pasted upon the posterior face of the lens. It should be recalled that any type of occlusion which is applied to the glasses frame or lens may not be fully effective because of peeking. Peeking, however, may often be avoided by the ophthalmologist putting one drop of atropine in the occluded eye

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daily as long as peeking persists, and terminating it when the peeking has stopped. Obviously, the higher the refractive error, the more effective is this atropine as a supplement to occlusion. The patient should have good and nearly equal vision before beginning dissociation treatment.

2. THE OVERCOMING OF SUPPRESSION

The next step in the treatment of accommodative esotropia is the eradication of foveal suppression. The child is placed on the major amblyoscope with the suppressing eye fixating a foveal dot or any foveal target. The picture is held stationary while the dominant eye fixates a circle into which the dot barely fits. The child is instructed to move the circle until the dot is immediately to the left side of it, and then move it to the right side. At first the dot will skip from one side to the other without going through the circle. When the patient has achieved the centering of the dot in the circle (superimposition), a foveal fusion target is placed in the major amblyoscope, and if there is no suppression, training of fusional amplitudes is started.

3. THE STIMULATING OF DIPLOPIA

Diplopia exercises are carried throughout the whole series of treatment. Before doing these exercises, the patient must be taught the difference between straight and crossed eyes. If, with correction, the patient is straight for near, the correction is then removed; if he wears bifocals, he looks through the top segment while he fixates a target 13 inches away. The reason for this is that the images will be closer together when looking through the top segment than they are without correction, thus making it easier to recognize double vision. A red filter is then placed over the squinting eye, and as the patient fixates the white light with the dominant eye and the technician covers and uncovers the eye behind the red filter, the red light begins to blink on and off. As the covering and uncovering is slowed down, he should appreciate homonymous diplopia. While he continues to fixate the white light, the red filter is moved on and off until he can hold diplopia without the red filter.

He should also be able to appreciate diplopia on a small picture with print. He must learn to recognize that at this point in his training clear vision means crossed eyes and blurred vision means straight eyes. When he has learned to appreciate diplopia with crossed eyes, confusion results, the diplopia is unacceptable, and he straightens his eyes to avoid it. If he has difficulty in straightening, plus three spheres are held in front of his eyes while he fixates a light 13 inches away. If he does not cross his eyes when the plus threes are removed, he will have blurred vision. He must learn to cross and straighten voluntarily. With his glasses off, the same procedure is done on a vision chart at 20 feet. To ensure cooperation, it is helpful to draw a picture of straight and crossed eyes and explain why it is necessary to see two things.

4. THE IMPROVING OF FUSIONAL VERGENCE AMPLITUDES

There are two types of fusional amplitudes to be considered; convergence and divergence where accommodation is not controlled, and relative fusional divergence where accommodation is controlled.

a. *Good fusional convergence and good fusional divergence with correction must be established.* In measuring this amplitude, the end point is diplopia. The convergence amplitude is of utmost importance. One cannot maintain straight eyes and do near work unless he has a normal fusional convergence. The child who squints has forgotten how to converge properly and has to be taught, or he may have squinted since an early age and has never used this function. If he is taught only to maintain an orthophoric position of his eyes at distance, and taught only to diverge, the orthoptist has gone only half-way in curing his condition. When he can diverge

approximately 5 diopters and converge approximately 40 diopters on the major amblyoscope, he is ready for dissociation of his accommodation and convergence.

b. *Relative fusional divergence.* When the patient can fuse a foveal target at or near zero on the major amblyoscope and read with clear vision when fully corrected, minus lenses should be inserted, —0.50 S at a time, and divergence to an end point, which is indicated by blurred vision or diplopia, should be stimulated. The same procedure may be followed for the reading distance by adding minus 3.00 S to each eye, recalling that the "zero setting" would be 20 prism diopters convergent for a 60 p.d. The telebinocular, stereoscope, rotoscope and similar instruments may be used in the same manner.

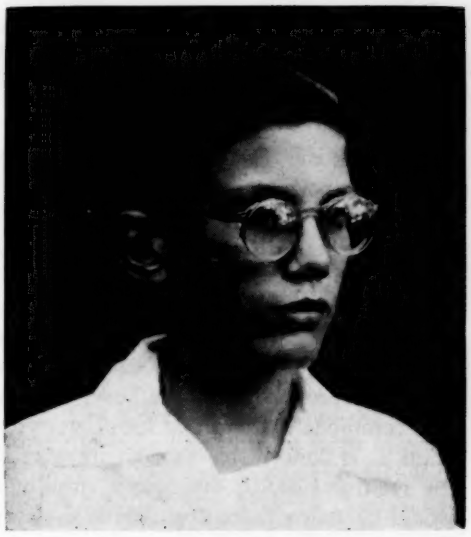

FIGURE 1.

5. DISSOCIATION OF ACCOMMODATION AND CONVERGENCE

The next step is to determine the minimal refractive correction which will give him single binocular vision while accommodating distance and near. This has been discussed by Dr. Costenbader, but I wish to emphasize that it would seem to be a mistake to prescribe +3.00 adds routinely instead of determining how little add is necessary to give the patient 20/30 vision with single binocular vision at near. When one is concerned with reducing glasses until the patient is able to have straight eyes without correction and with clear vision, he must be aware of the importance of allowing as much accommodative effort as necessary while maintaining straight eyes. It is just as important to give the minimal amount of correction which will allow single binocular vision and 20/20 at 20 feet.

In treating accommodative esotropia, we are increasing the amplitude of relative fusional divergence. In order to eliminate changing the patient's glasses frequently as he learns to increase his relative fusional divergence, minus fit-overs are used in increasing strength until the glasses have been neutralized and the eyes are straight without glasses while accommodating. This, of course, must be done only with permission from the referring ophthalmologist.

If the patient can read 20/20 with correction with straight eyes, a —0.50 S fit-over is placed over his glasses (fig. 1) and his state of binocular vision is determined while accommodating on the chart at 20 feet. If with this —0.50 S he can read at least 20/50 or 20/40 with straight eyes, this strength fit-over is given for constant wear as well as for home exercises on the chart. Within a few days he will probably have single binocular vision with 20/20 vision. When he can read 20/20 with the —0.50 S and keep straight eyes, fit-overs are progressively increased in strength until the glasses are neutralized. The same procedure is continued until he is using the highest strength minus lenses that he can tolerate without asthenopia or strabismus and with 20/20 vision. This represents the outer limit of the relative fusional divergence. In the event the patient is wearing bifocals, there is a simultaneous reduction. The strength of minus lens that will allow him 20/20 vision with single binocular vision at near is given, until the bifocals are neutralized. Then the bifocals are re-

moved if pasted on, or covered if fused, and the same procedure carried out until he can read 20/20 without correction with straight eyes at near also.

At times the distance correction reduces out of proportion to the near correction, and at other times hand in hand. Those children whose distance correction reduces out of proportion to the near correction have an abnormal accommodation-convergence ratio. Since such an abnormal ratio is manifested while accommodating at near, all out of proportion to the lesser amount of accommodation required for distance fixation, it is conceivable that an increase in the relative fusional divergence amplitude would be much more apparent at distance than at near fixation.

Those children whose distance correction reduces hand in hand with the near correction have a normal accommodation-convergence ratio. As they can handle greater amounts of hypermetropia on their own with the improvement orthoptics has given them, their improvement is manifested equally at distant and near fixation.

In the event the distance correction reduces out of proportion to the near correction, it is necessary to give two pairs of fit-overs, one for outdoor play and one for close work. May I emphasize that it is not advisable to increase the minus fit-overs more than one-half diopter at a time since several days are necessary to stabilize the new amplitude of relative fusional divergence. With the minus fit-overs worn at home the progress is much faster. In six weeks, many children with hypermetropia of +2.00 to +3.00 will completely dissociate; others take three to six months.

In many cases in my experience, the amplitude of relative fusional divergence increases to a point which allows single binocular vision of about 20/70 without glasses for distance and near, but will not increase further. A longer period of time is required on each step of increasing the minus fit-overs and some cases do not respond even with longer periods of time; however, we have some hope for these cases, as Dr. Costenbader has pointed out, with the use of DFP in allowing these patients to progress further.

To make the patient doubly aware that his eyes are straight and fusing during the procedure just described, physiologic diplopia exercises are given, such as framing, hole in hand, the Walraven separator, and many others to be described by Mrs. Kramer.

The metronoscope is excellent for developing relative fusional divergence. As the child reads, base-in prisms are increased to the end point of diplopia or blurred vision. Whatever strength of prism he can overcome in the office is given to him for home bar reading.

For a month after the patient has obtained his maximum result, the intensive work should be carried on at home to stabilize the new relationship between accommodation and convergence. He should be seen once a month in the office for several months to check on his ability to maintain straight eyes without glasses or with reduced correction.

Symposium: Accommodative Esotropia

ORTHOPTIC TRAINING AT HOME

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THE IMPORTANCE of home training cannot be overemphasized in the treatment of accommodative esotropia. Exercises must be performed daily to effect a cure, and since it is impossible, in the average case, to see the patient in the office or clinic every day, the burden of the exercises must be placed on the parents and the child. In most cases this role falls to the mother. She must therefore be made to understand that exercises must be carried out daily and must be fitted into the schedule of family life. Explicit instructions are given to the mother regarding each type of current exercise. It is not enough to hand her a mimeographed sheet of instructions on each exercise; this is indeed helpful, but she needs to see just how the ophthalmologist or orthoptic technician achieves the desired result. To this end, it is desirable to have the mother present in the treatment room the day the new home exercises are explained to the patient.

Many of the ophthalmologists in our audience may not have the services of an orthoptic technician available but would still like to be able to prescribe home exercises for the accommodative squinter. To these physicians, I would suggest that mimeographed copies of instructions for home training be given to the parents, emphasized by a concise explanation. The intelligent parent will be able to carry out most of these exercises without taking too much of the doctor's valuable time.

AMBLYOPIA TRAINING

Total occlusion and the various types of lens occlusion have been discussed in the preceding paper. The wearing of the occlusion, however, should be augmented by all forms of near work. This is especially important in the preschool child of the type who prefers to play outdoors all day long and whose toys are so large that they do not stimulate macular and foveal vision. The mother of this type of child should set aside an hour in the morning and an hour in the afternoon for crayoning, bead work, cutting pictures and other entertaining tasks of this nature.

When the suppression becomes foveal only, and the vision has improved to within one line of the better eye, strip occlusion on the nasal side of the lens before the good eye seems to give better results than other types of occlusion. Music mending tape or Permafilm is used. This prevents use of the good eye when the patient is converging for near tasks and permits binocularity whenever the visual axes are in the primary position. Thus, fusion is being stimulated and at the same time foveal suppression for near is being minimized. A red filter applied to the lens before the good eye is also helpful in stimulating vision and breaking up foveal suppression, as Miss Tibbs has described. It is not necessary to use strip occlusion in the majority of cases where foveal suppression exists so long as the child is actively engaged in treatment in the clinic, as well as carrying out antisuppression exercises at home.

ANTISUPPRESSION EXERCISES

Preschool Age

A red glass filter is placed before the good eye. Exercises are performed, such as sewing with red thread or yarn; jig-saw

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puzzles, construction paper-work; stringing small seed beads; crayoning and tracing with red pencil. Since the red filter will obliterate the colors yellow, orange, pastel shades and white, many games can be utilized. Remember to place the red filter before the nonsuppressing eye.

School Age

A red filter is placed before the fixating eye. Stories can be typed in red and black print by the parent or willing friend for this exercise; the Dobson reader is also useful. Sewing and embroidery work are enjoyed by girls. Gimp and Indian-bead loom work is given to both boys and girls. The bead loom work seems to be the best exercise we have at our command for eliminating foveal suppression, since the beads are very tiny and require foveal fusion. Boys are just as enthusiastic about it as girls, perhaps more so, since this particular type of handicraft is a part of the Boy Scout program. Therefore, Indian belts, watch fobs, wrist-watch bands and bookmarkers are exhibited with great pride and a sense of achievement. To stimulate interest in home training, many clinics and offices utilize a bulletin board where varied types of handicraft are exhibited for all the patients to see. School children who have much home work are urged to wear the red filter and write their spelling and arithmetic assignments with a red pencil. I have yet to find a teacher in any of our public, private or parochial schools who will not accept home work in red pencil when the reason is explained.

The small American-made cheiroscope for tracing can be prescribed at any age and does much to remove suppression. The red filter is not worn while performing this exercise.

As Miss Tibbs has explained, diplopia exercises are taught in the office and carried out as a home exercise. Although a simple exercise, it is not always thoroughly understood, so that for purposes of emphasis, the technic is reiterated. The child's correction is removed and a red filter is placed before the squinting eye. He is taught to squint voluntarily, appreciating homonymous diplopia and maintaining it. A great many patients will cross the good eye when asked to perform this act, since they do not want to relinquish the suppression reflex they habitually manifest when the squinting eye is turned in. If the good eye is permitted to cross to achieve diplopia, nothing has been gained in eliminating suppression of the squinting eye. Occasionally, it takes two to three weeks to teach a child to cross the squinting eye and appreciate diplopia on a light. This exercise is performed on various lights at home, i.e., ceiling lights, lamps, television, small and large flashlights, both at distance and near. As soon as the child can get immediate diplopia on a light whenever he wishes, the red filter is removed and diplopia is sought looking at objects of all sizes and at varying distances.

When diplopia is firmly established in the squinting act, physiological diplopia is taught. At first the patient wears his glasses to perform this function and uses a pencil to "frame" objects. By "framing" we mean that when fixating an object such as picture on the wall, he can appreciate two pencils. By varying the distance of the pencil from the eyes he can place the two images so that they appear at the edges of the object fixated. He is then given bar reading with the standard bar reader or a cardboard separator made from a piece of stiff cardboard with a tongue depressor affixed to the edge farthest from the eyes. These can be made within a few minutes.

FUSIONAL AMPLITUDE

If the fusional amplitude with the glasses is deficient, a hand stereoscope is prescribed, using cards with a base-in effect for divergence and base-out effect for convergence. Some of these charts are the Keystone, Dvorine and Wells sets. Home-made charts can easily be made utilizing two stickers placed on a piece of cardboard with sticker stars or dots for control markings.

DISSOCIATION OF ACCOMMODATION AND CONVERGENCE

As soon as the fusional amplitude is normal with glasses, exercises to dissociate the convergence and accommodation are begun. A visual acuity chart for home exercise is prescribed. Large uncluttered pictures are substituted for a visual acuity chart for the preschool child and can be set up 20 feet from the patient. Very small pictures with foveal detail are used for near and are easily handled if pasted on a tongue depressor. For the school child who can read, the advertisements sheets in the newspaper can be used. The child practices accommodating without glasses, i.e. clearing his vision, while inhibiting his excessive convergence, with resultant straight eyes. The goal is to read smaller and smaller print. This exercise is performed not only in the house on the pictures, visual acuity chart and printed matter, but is executed outdoors using street-signs, license plates, signboards and advertisements on trucks. He is instructed that there is no time limit on this exercise and that the more he practices the sooner he will begin to clear his vision while keeping eyes straight. The child who constantly asks "How long must I do this exercise every day" is the child who makes slow progress. If the ophthalmologist and the orthoptic technician can stimulate the patient to try this at every available opportunity during the day, he is soon rewarded by clarity of vision with weaker or no glasses.

As it has been stated in the three previous papers, the patient who can maintain single binocular vision for near with his distance correction, does not need a bifocal and has a normal or nearly so amplitude of relative fusional divergence. Home instruments that can be prescribed for cases that fall in this category are:

For distance use Stereoscope or Correcteyescope, View Master, and True-View. For near use Stereoscope or Correcteyescope, Walraven Bar Separator, Remy Separator, and Diploscope.

Cases requiring bifocals have an abnormal accommodation-convergence ratio because they have inadequate relative fusional divergence. Therefore this type of patient needs special home exercises at the near point to develop this function. It is generally unnecessary to prescribe exercises for distance. Home exercises at the near point are outlined as follows:

Stereoscope. Set at near point and leave there so that the convergence is fixed. Use Keystone Delta, Alpha and Gamma base-out charts, Dvorine, Wells One and Target sets. Start with the maximum base-out chart in the series and work gradually to the orthophoric chart. This stimulates the relative fusional divergence.

If the Correcteyescope is used, split slides can be employed at the near point. There are many reading charts, graded as to visual acuity, which can be obtained.

Orthofusor base in. The Orthofusor base in should be placed at a fixed distance from the eyes. It can be propped on a table in front of the patient, with instructions to him not to vary the distance from his eyes. The distance must be fixed because the change in relative fusional divergence is induced by the disparity of the images on each succeeding picture. If minus fit-overs are used while performing this exercise, it increases the relative fusional divergence.

Orthofusor base out. Converging exercises are given in this exercise while maintaining the proper amount of accommodation for single binocular vision.

Walraven bar separator. This is a device for near work which makes separator reading more comfortable since the reading material is placed on the instrument at a fixed distance. With the minimal correction necessary to hold the patient's eyes straight while accommodating for near, he must be conscious of the two separators (i.e., physiological diplopia) while he fixates the center of the page. A set of cards for practicing steady single binocular vision comes with the instrument. Unless the fusion is secure, the patient will alternate;

Consequently the parent and the child must be attentive and alert during this exercise. One of the cards that is supplied with the instrument is worth mentioning. You will note that the card shows 17 A's. The patient points to the A in the center which appears in the middle of the two separators if he is maintaining fusion. By counting from right to left, adding only one A at a time, he goes into fusion and out of fusion as he moves away from the center. He learns to do this until fusion is instantaneous when he is in the fusional area, thus overcoming diplopia or suppression. When he can perform this exercise, he is ready for the same exercise on fine print. A page from a printer's type catalogue is given him for home use. He is then ready to graduate to reading books and writing on the separator. School home work such as spelling and arithmetic can be performed on the Walraven Bar Separator.

Minus fit-overs. For those who cannot immediately dissociate the convergence and accommodation and thus remove glasses, minus fit-overs are prescribed by the attending ophthalmologist. The orthoptic technician can keep the ophthalmologist in charge informed as to how much reduction in strength of his correction the patient can take and still maintain single binocular vision and have clear vision for distance and near. It has been our policy in Washington to have in our offices and clinics, fit-overs of —0.50 sph. to —3.00 diopters, in half diopter increasing strengths. It is often possible to increase the strength of the minus lenses 0.50 D. every ten days to two weeks. Sometimes there is an overestimation of the patient's ability and on the next visit the fit-over has to be changed to a weaker strength. The purpose of the fit-over is to increase the patient's ability to exercise his amplitude of relative fusional divergence. It also has the added advantage that the correction does not have to be changed nearly so often, thereby eliminating unnecessary expense to the patient.

The child wears the fit-overs all day. This speeds up his ability to accommodate so that even during school hours he is being exercised. Let me emphasize the fact that diplopia in the squinting act must be an established function before minus fit-overs are prescribed; otherwise, he might squint with the reduction in his correction and suppress, and not know that his eyes are crossed. With the constant threat of diplopia over him, he learns to maintain single binocular vision and use his relative fusional divergence.

If the devices and instruments mentioned are used daily, the treatments at the office or clinic can be markedly reduced. The length of treatment to effect a cure is determined solely in most cases, by the cooperation of the parent and child in carrying out the home training with enthusiasm. It has been our experience during the last year that since the use of the minus fit-overs has been added to our armamentarium, the length of treatment to cure an accommodative esotropia has been reduced by many months.

As Dr. Costenbader and Miss Tibbs have emphasized, not all our cases are easy ones. Difficult conditions are encountered such as the child with the remote punctum proximum possibly due to constitutional factors; borderline fixation disparity cases which probably never do secure bifoveal fixation; the bifocal cases which have little or no refractive error for distance and whose relative fusional divergence is resistant to any of the exercises outlined; and the cases of heterophoria with divergence insufficiency that almost attain the goal and then suddenly regress. However, despite the disappointments, the larger number of cases we do help and cure make the effort involved seem very worth while, and stimulate us to still further research in our solution of the more difficult cases.

ANATOMIC CONSIDERATIONS IN THE DIAGNOSIS OF STRABISMUS

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IN THE analysis of the various oculomotor problems a knowledge of the anatomic background is fundamental. In this presentation, certain anatomic data will be presented which are the basis for abnormal muscle action and a knowledge of which is essential to a correct analysis of many oculomotor problems. The discussion will be limited to but one phase of the subject; namely, anatomic considerations of the peripheral oculomotor mechanism.

The peripheral oculomotor mechanism may be divided into two parts, the vertical and the horizontal extrinsic muscles and their respective surrounding fascial structures. It is important to appreciate the fundamental anatomic and physiologic differences between the vertical mechanism and the horizontal mechanism. Not only do they have separate brain centers but the peripheral mechanism is fundamentally different. Diagnostic interpretation demands an appreciation of this fact.

Anatomic and physiologic evidence points to a factor of stability in vertical muscle balance. The integration of the various types of muscle action (vertical, horizontal and torsional) which is present in varying degrees as the gaze is shifted from place to place necessitates a stable vertical mechanism. The arrangement of the four vertical muscles represents the most economical way in which to achieve both a vertical movement free of torsion, following Listing's law, and a torsion of the eyeball where this is needed.

This stable vertical balance seems essential in the binocular act, permitting the horizontal component to retain the high degree of flexibility necessary for accommodation and convergence.

Certain data indicate the stable nature of the vertical mechanism.

1. Stability of the vertical muscles is present at birth; the newborn possesses the power of conjugating the eyes in vertical movements. In contrast, horizontal movements are poorly conjugated at birth and require months before becoming stabilized. Indeed, full stabilization usually does not occur for several years, and even then the horizontal movements retain a variability which permits a wide range of flexibility and may under unfavorable conditions easily lead to instability.

2. Embryology of the vertical muscles points to a factor of stability. A study of the developmental stages shows that a definite pattern in the arrangement of the vertical muscles is present at the earliest stages and corresponds fundamentally to that of the adult.

3. Comparative anatomy also emphasizes that stability of the vertical balance is essential to efficient binocular action. In the lower forms the ocular movements are simple. In man, due to environment the vertical mechanism is complex, owing to the variable actions depending upon the direction of gaze, whereas the actions of the horizontal muscles are comparatively less complex.

4. The vertical mechanism has a very limited range of reflex power of adjustment, while the horizontal component is endowed with great power of adjustability.

5. A disruption of the vertical balance produces a definite mechanical obstacle even though it is small in amount.

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With this brief review of some of the factors relating to the normal mechanism, I wish to discuss briefly a few of the abnormal peripheral anatomic factors and their relation to abnormal oculomotor phenomena encountered in diagnosis. These peripheral defects create a definite mechanical obstacle to binocular unity and are a basic cause of muscle imbalance.

PERIPHERAL DEFECTS

Clinical Manifestations

Like other infranuclear involvement, peripheral motor defects show certain prominent characteristics which identify them.

1. The resulting deviation will relate to the action of individual muscles and will therefore be noncomitant in type. A deviation which is usually present in the primary position increases or decreases in different parts of the field of fixation depending on the muscle involved. Because of this fact the version test, in which binocular movements are tested, is of great value in analyzing the problem.

2. In addition to the noncomitant nature, the uniformity or constancy of the angle of the deviation is of value in identifying the defect.

3. Additional proof that the anomalous position of rest is based on infranuclear conditions may be obtained by excluding, as far as possible, those innervations which may affect the position of the eyes relative to each other; for instance, by correcting any existing hyperopia and suspending the influence of the fusion apparatus. Also the nature of the deviation when the patient is under anesthesia is suggestive. The deviation which persists under these conditions is largely mechanical.

Cause

A peripheral anatomical defect may be caused by an abnormal state of the muscle structure, an abnormal mechanical relationship of the origin or insertion of the muscle, or an abnormal relationship of the fascia (due to maldevelopment or to a mechanical factor such as trauma).

These mechanical factors are apparently much more common than is generally recognized. There is substantial evidence to indicate that an appreciable percentage of the peripheral defects are mechanical and not paretic in origin. Scobee[3] states that in 90 per cent of all patients with heterotropia which develops before the age of 6 years there is some underlying mechanical anomaly.

Such defects will cause a muscle to underact, so that findings at examination may simulate a paresis of the muscle. (The word "paresis" infers that the basic cause for the abnormal action is a nerve involvement. Some consider that most cases of nonaccomodative strabismus are paralytic in origin and refer to these underacting muscles as paretic. However, such a conclusion is not substantiated by the evidence. A certain number of defects are of paretic origin; in some instances this origin is evident, but in the majority of cases the evidence of paresis is not conclusive. As the term is rarely based upon actual knowledge of a defect in innervation, it conveys an erroneous impression. In referring to restricted action of an ocular muscle, the term "underaction" expresses the situation more correctly. It is preferable in the absence of knowledge that the cause is actually an involvement of the nerve to the affected muscle.)

An anatomic anomaly may not in itself be sufficient to produce heterotropia. Innervational impulses neutralize the effect of a high percentage of such abnormal mechanical situations. However, there is a limit to the adjustment capacity of the binocular mechanism. When taxed beyond its capacity, deviation results. If the effect of the defect is slight, the result is only heterophoria. If the effect is greater, the tendency toward heterotropia results; some other factors—accommodative, innervational—may precipitate the heterotropia.

Major defects are readily recognized because of the binocular problem which is usually present. Slight defects are frequently not evident and are discovered only on careful examination. However, minor defects are of considerable importance because they may be a factor in the development of squint at a later date, if for some reason binocularity fails to function.

The defects caused by noninnervation peripheral factors may be grouped into the following categories: (1) defects related to the eyeball; (2) defects related to the orbit; (3) defects related to the fascia, and (4) defects related to the muscles.

Defects Related to the Eyeball

These factors are found chiefly in horizontal defects and are not as a rule primary factors in vertical defects. Developmental obstacles (anisometropia, etc.) prevent an adequate establishment of binocular vision during the period when the binocular reflexes are normally conditioned. Pathologic obstacles affect the formation of clear images, especially if the opacities are unilateral or unequal on the two sides.

Defects Related to the Orbit

The topographical relationships between the eyeballs and their adnexa are of importance in determining the position of the eyes and the freedom of their movements. Positional obstacles may be developmental, due to the anomalies in the symmetry and inclination of the orbits, in the shape of the skull, in the shape of the globes themselves (particularly in high myopia) and in the position of the globes (as in proptosis); due to disease (inflammatory, neoplastic) displacing the eye; due to orbital injury resulting in damage to the tissues which support the eyeball, or to organization of blood clot interfering with the action of the muscles.

All these obstacles may considerably impede the free excursion of the eyes and disorient the pull of the muscles so that binocular action is impaired.

It is important to remember in discussing this group of causative factors that, as long as fusion is normal, an imbalance may not exist even with a definite difference in the level of the orbits or other factors tending to create lack of alignment of the eyeballs.

Defects Related to the Fascia

Fascial abnormalities may be acquired or developmental. The acquired fascial defects may result from scar tissue formation following injury, inflammation or a deviation of long standing. An eye deflected for many years may acquire fascial contractions conforming to the fixed position of the globe. A check ligament not called on to perform its function for a number of years may lose some of its elasticity and thus act as an obstacle to outward rotation. Some question the importance of the time factor in the development of abnormal check ligaments, because the same anomalies are found in the 2-year-old and in the 30-year-old with esotropia.

An abnormal development of the fascia which surrounds the extrinsic muscles may cause a mechanical defect and produce a deviation. If the defect is slight, the patient may pass through the critical fusion-development stage and the resultant deviation tendency is then kept latent as a phoria. If the defect is pronounced, fusion does not develop and a tropia results. In developmental fascial defects the anomalies precede the tropia in point of time and the tropia is at least partly due to the anomaly.

Abnormal development of the fascial covering of the extrinsic muscles and the check ligaments is believed by some to be more common than the textbooks indicate, exerting a definite influence on the action of the binocular apparatus. A series of dissections will demonstrate marked variation in the fascial development.

Anomalous fascial formations may be explained on the basis of incomplete maturation of the mesoblastic tissue. Both muscle and fascial tissues originate from

the same mesoblastic tissue; muscle tissue represents a full maturation of the mesoderm; the fascia which surrounds the muscles and tendon represents mesoderm which did not proceed to that full degree of differentiation. A variation from its usual degree of maturation might possibly produce an abnormal fascial development. This abnormal development could influence the normal action of the muscle by producing, for example, a defective check action.

Abnormal fascial formations associated with the horizontal muscles are quite frequently observed. Because of this accessibility, such abnormal fascial formations are readily detected.

Scobee[3], in his description of abnormal fascial conditions and how they affect horizontal muscle action, points out that certain types of muscle imbalance can be attributed to abnormal fascial development. He found in esotropia the following variations from normal fascial development existing either separately or in combination.

1. Extra and thickened check ligaments. The normal main horizontal ligament is present, quite obviously thickened and somewhat contracted. Extra check ligaments arise from a line paralleling the origin of the main ligament, either just above or just below it.

2. Fused check ligaments. Three or five ligaments are apparently fused together into a thick solid mass running from the muscle sheath to the orbital wall.

3. Posterior check ligament. A posterior check ligament arises from the muscle sheath far back in the orbit and runs anteriorly, inserting into the medial orbital wall along its entire course.

The anomalies just listed were found by Scobee in more than 52 per cent of all horizontal tropias which had appeared before the age of 6 years and which were known not to be due to trauma or systemic disease.

The existence of any one or all three abnormal fascial developments, Scobee believed, will prevent proper movement of the globe with a muscle hook under general anesthesia, and may cause retraction of the eyeball if rotation of the globe is forced.

Scobee's observations have been confirmed and it is generally accepted that a variation in fascial formation is a positive factor in creating abnormal action of the horizontal muscles in an appreciable number of cases.

Although Scobee's work emphasizes the fascial abnormalities of the horizontal muscles, it is possible that overaction and underaction of the vertical muscles can be explained on the same basis. A study of the complex anatomy of this area suggests the feasibility of such an explanation.

That fascial anomalies are the cause of abnormal action of the superior oblique is a debatable point; we have very little definite evidence to substantiate such claim. An abnormal fascial development or check action is difficult to demonstrate during operation on the superior oblique; it is also difficult to demonstrate in a dissection because of post-mortem changes. It is, of course, conceivable that such anomalies exist, but the degree to which they influence the action of the muscle can only be surmised.

The author has encountered cases in which the fascial membranes surrounding the superior oblique tendon showed definite evidence of contraction and caused a disturbance in the action of the muscle. In some instances, the cases were associated with a marked underaction of the inferior oblique and, as described by Brown[1] in his presentation of the superior oblique sheath syndrome, may have been secondary to a defective inferior oblique. However, in some cases the inferior oblique muscle seemed structurally normal, and the sheath of the superior oblique was congenitally short.

In other cases a mechanical factor—for example, direct or indirect trauma such as results from surgery on an adjacent tissue—disrupts the fascial relationship. Thus, a re-

cession of the superior rectus can cause adhesions which interfere with the action of the adjacent tendon of the superior oblique. Likewise, extensive resection or recession of the medial rectus can interfere with the fascial sheath of the adjacent superior oblique.

In the case of the inferior oblique we have more evidence of abnormal fascial formation, both at the time of surgery and in the dissecting room. For example, the author has observed thickened fascial bands which unite the inferior oblique and lateral rectus sheaths more intimately than usual. Also the posterior fascial expansion of the muscle sheath may be abnormally developed or contracted. Both of these abnormal fascial formations restrict more or less the normal action of the inferior oblique muscle. A series of dissections will demonstrate marked variation in the fascial development at the point of crossing of the inferior rectus and inferior oblique, with variable degrees of fusion. These variations in fascial formation might well have definite clinical significance.

Trauma resulting from injury to the inferior oblique muscle or surgery on the lateral or inferior rectus may create scar tissue which will restrict the action of the inferior oblique.

Spastic overaction of the inferior oblique is seen frequently. Although various explanations have been given for an overacting inferior oblique, abnormal fascial development may be considered a possible cause.

Like the obliques, the vertical recti can be inhibited in their normal action by abnormal fascial conditions, either aacquired or developmental. The acquired defects may result from trauma, such as a blow or perforating injury, or from scar formation following surgery on these or adjacent muscles. Developmental defects of the fascial coverings and check ligaments account for some of the abnormal action of the rectus muscles. Like the superior obliques, the vertical recti are inaccessible, and, because of their complex fascial coverings abnormal development is difficult to recognize. Certain evidence, however, demonstrates its presence. For example, the so-called vertical retraction syndrome mentioned by Brown is an example of a congenital maldevelopment involving the vertical recti. Brown considers the condition due to congenitally paralytic superior and inferior rectus muscles of the same eye, and suggests the presence of limiting fibrous bands in this area. Of the 10 or 12 cases that Brown has observed, all but one showed retraction movement on elevation only. Brown states that the retraction movements in this anomaly are not so evident as those in the retraction syndrome, but on maximum effort of monocular elevation of the affected eye in the temporal field there is a definite retraction with some narrowing of the palpebral fissure.

Defects Related to the Muscle

They may be nondevelopmental or developmental in origin.

I. Nondevelopmental Defects

Trauma to a muscle.—Usually the injury is slight, such as rupture of the sheath or a hemorrhage into the body of a muscle, leading to little or no damage. At times it may be pronounced and produce permanent fibrosis.

Myasthenia gravis.—The paralysis may involve various eye muscles, although the intraocular muscles always remain exempt. Characteristic of this paralysis is a rapid increase in degree when exhausted. The diagnosis is established if there is associated feebleness of muscles of the face, neck and masticatory apparatus. The prostigmine test is of value.

Hereditary ophthalmoplegia externa.—This condition, which may not become obvious until adult life, has been explained as an abiotrophy of the muscle fibers concerned.

Tumor.—A neoplasm may originate in a muscle; for instance, a fibrosarcoma; or the muscle may become involved by malignant mestastases.

II. Developmental Defects

Defects of congenital or developmental origin are important because defective cleavage of the mesodermal sheet from which the muscles develop is probably much more common than the literature would indicate. Minor anomalies which can be compensated by the corrective fusion reflexes (abnormal insertion or weakness of a muscle) probably cause some cases of heterophoria; greater degrees which cannot be thus compensated lead to a squint in which there is incomitance.

As to frequency of ocular muscle anomalies, Whitnall[4] states:

> It is probable, to judge from the writer's individual experience in finding quite a number of gross abnormalities of the ocular muscles in a series of dissection, that such are by no means as excessively rare as would appear from the number recorded in the literature; dissecting room conditions do not favor their identification, and in life some may be unrecognizable through compensatory action of the other muscles.

Clinically, anomalies are not frequently observed, although possibly often overlooked, as suggested by the frequency with which they are encountered in the dissecting room. Many cases of congenital squint are apparently dependent upon such anomalies, and it is only by an appreciation of this fact and a better knowledge of these peculiarities that the surgeon can anticipate the presence of an anomaly before operation.

Developmental anomalies of the muscle may be considered under the following heads: A. Anomalies of the muscle; B. Anomalies of the insertion; C. Anomalies of the plane of action of the muscle.

A. Of the Muscle

Absence.—Judging from the number of cases reported, total absence of a muscle is exceedingly rare.

Casten encountered congenital absence of the inferior rectus muscle and was able to find 10 other cases reported in the literature. McDonald described the absence of the inferior rectus muscle (proved by operation). Posey reported the inferior rectus muscle absent except for a rudimentary portion at the usual site. Stieren described absence of one, and Davis absence of both inferior rectus muscles.

Steinheim reported a case of absence of the superior rectus muscle. Natale reported a case of congenital strabismus in which the superior rectus and inferior rectus muscles were found absent at operation. Coover, in operating on a woman in an attempt to advance what he thought were paralyzed superior and inferior rectus muscles, found them absent.

Krause described the absence of the medial rectus muscle. Klincosch reported absence of all the ocular muscles. A few cases of the absence of oblique muscles have been reported.

The above reports, representing but a portion of the literature, serve to emphasize that one, several, or all of the extraocular muscles may be absent. Furthermore, after reviewing the literature, Posey concluded that anomalies of the ocular motor muscles are more frequent than published reports would indicate.

Fibrosis.—Muscles are frequently found to be relatively inelastic. A muscle that is constantly prevented from relaxing adequately may eventually become somewhat contracted and later slightly fibrotic. For example, the so-called overfunction of the inferior oblique may be a secondary contracture, following paralysis of the superior oblique of congenital or early origin. The degree of fibrosis is highly variable. In some cases the muscle is practically replaced by a fibrous band (Duane's syndrome, where the squint is due to a contracture in a muscle).

Abnormal development.— This condition is more frequent than absence or fibrosis of a muscle.

Horizontal rectus muscles.—A definite fusion between the lateral rectus and the inferior oblique is occasionally seen at operation. The author has had 2 such cases. Bundles of muscle fibers from the lateral

to the inferior rectus, to the orbital wall, and to the lower lid are described by Whitnall. A lateral rectus with two extra fasciculi which passed forward to end on the inferior tarsal plate and lateral wall is recorded by Curnow. In a specimen Whitnall found a fleshy bundle 7 mm. long and 2 mm. in diameter, passing from the lateral rectus across the posterior third of the orbit beneath the optic nerve to fuse with the belly of the medial rectus; no nerve could be traced.

Although abnormal formations of the medial rectus undoubtedly exist, the literature yields but few reports. Those cases reported were related to the degree of muscle development rather than abnormal fusions.

Vertical rectus muscles.—A developmental tendency to anatomic abnormalties in the vertical recti is seen chiefly in the presence of supernumerary slips of muscle tissue. Abnormal muscle slips may rise well back in the orbit and diverge slightly from the course of the muscle which is their apparent source to become inserted into the globe at a point somewhat behind and to one side of the insertion of their parent muscle.

The above characteristics, representing but a portion of the literature, show that one, several, or all the extraocular muscles may be involved in abnormal development. Also, it seems apparent that anomalies of the ocular motor muscles are more frequent than published reports would indicate.

Superior oblique muscle.—The muscle may end at the trochlea, or a normal superior oblique may be accompanied between the eyeball and the pulley by an extramuscular slip which has a common insertion with it upon the eyeball.

The superior oblique may be closely accompanied by an off-shoot from the levator palpebrae superioris, sometimes called comes obliqui superioris.

Ledouble found supernumerary fasciculi accompanying the reflected tendon.

According to Prangen two instances have been found in the human in which the superior oblique muscle ended in a common tendon with the terminal nasal fibers of the superior rectus muscle. This type of anomaly very likely is more frequent than reports in the literature indicate.

Inferior oblique muscle.—Some variation in the point of origin of the muscle has been observed.

An abnormal muscle bundle (musculus obliques accessorium inferior) is occasionally observed. Rex reported an abnormal muscle bundle passing from the apex of the orbit to the inferior oblique, also sending a slip to join the inferior rectus; the anomaly was found in both orbits and was supplied by the third nerve.

An important anomaly which involves both obliques is referred to as "voluntary propulsion of the globe."

The point of crossing the inferior oblique and the inferior rectus shows considerable variation in the degree of fusion; some specimens are firmly, others loosely, fused. These fusions appear to be fundamentally fascial in character. Abnormal fusion at the point of insertion of the inferior oblique has been noted quite frequently. This is for the most part with the lateral rectus muscle and in some cases appears to be both fascial and muscular in character.

B. Of the Muscle Insertion

Abnormal insertion of the extrinsic muscles is a mechanical etiologic factor which may exist in the peripheral mechanism.

Horizontal recti.—Because of their accessibility, variation in the insertion of the horizontal recti have been occasionally observed. The following illustrate some of the variations.

The lateral rectus may be inserted into the globe by two tendons. Posey reported a lateral rectus inserted into the globe by two tendinous bands, symmetric in size and development, and about 4 mm. apart. The tendons blended into two well-developed muscular bellies, which ran parallel

to one another until they were lost posteriorly in the tissues of the orbit.

The case just described is somewhat analogous to Wicherkiewicz', who found, while performing an advancement of the lateral rectus, that the muscle was inserted by two tendons, separated vertically by a distance of 4 mm. Dissection revealed that this space decreased posteriorly so as to form a triangle, with its apex in the belly of the muscle, 16 mm. from the line of insertion.

Macalister observed a lateral rectus supplied with two heads; Dieffenback, Behr, Zagorsky, and Albinus found double lateral rectus muscles.

In a case of retraction of the eye, Turck noted absence of the lateral rectus. The medial rectus was inserted 12 mm. from the corneal limbus, and also by a second head still more posteriorly, which accounted for the retraction.

In correcting a case of right convergent squint of high degree, Posey discovered an accessory muscle just inferior to the lateral rectus. This muscle, which was inserted into the globe by a short, rounded tendon 4 to 5 mm. long, immediately below the insertion of the lateral rectus, was well developed, more or less rounded, and took an oblique course inferiorly, midway between the lateral rectus and inferior rectus muscles, until lost in the tissues of the equator of the globe.

Peter reported an anomaly of the tendon of the lateral rectus. The muscle was unusually large and its attachment spread out in a fan shape, extending up to the insertion of the superior rectus. Division of the entire insertion corrected the deformity and restored the eyes to parallelism.

In advancing the left lateral rectus, Bourgeois found no trace of it at its usual insertion, but discovered it inserted far superiorly and in an oblique direction. The muscle was much atrophied, scarcely 2 mm. broad.

Bernheimer found a thin band of connective tissue in place of the lateral rectus. Axenfeld reported a similar case, and other instances of replacement of the lateral rectus by a more or less inelastic cord have been reported.

Similarly, the medial rectus shows a variation in its insertion although this variation deals chiefly with the length and position rather than a multiple type as found in the lateral rectus.

Vertical recti.—Very little data are available concerning abnormal insertion of the vertical recti. In the author's experience there is less variability than in the obliques. However, a certain amount of variation exists in the line of insertion, but insufficient data exist to make a positive statement.

Superior oblique.—The insertion of the superior oblique tendon is the most variable of all extrinsic muscle insertions. This marked variability is dependent chiefly upon two factors, namely, the variable length of the superior oblique insertion and the variable angle of its insertion. In specimens examined by the author certain variations of the insertion were so marked that abnormal actions of the muscle might possibly result.

Inferior oblique.—The insertion of the inferior oblique, though variable, is more uniform than that of the superior oblique. The variation consists chiefly in the degree of obliquity and convexity of the curve. The insertion often shows gross irregularities, such as angular serrations or dehiscences.

Vestiges of the musculus retractor bulbi (retractor obuli chanoid muscle) have on rare occasions been recorded in man. Ledouble found delicate muscle bundles in two cases and other instances have been noted by Nussbaum and Fleischer. According to Duke-Elder, it is always represented in man (30 specimens examined) by a well-marked strand of connective tissue (fascia retrobulbaris) lying between the lateral rectus and the optic nerve, and attached to the back of the fascia bulbi anteriorly; it does not appear, however, to be readily identifiable.

C. In the Plane of Action

Abnormal plane of action of a vertical muscle should be considered as a mechanical defect of the peripheral mechanism. This factor applies chiefly to the oblique muscles.

The abnormal planes of action of the obliques can be best appreciated by comparison with the normal planes. After passing through the trochlea the superior oblique muscle turns downward, outward and backward at an angle of about 54 degrees from its previous course, and becomes inserted into the posterolateral aspect of the globe, making a very small arc of scleral contact.

The inferior oblique muscle passes laterally backward and upward beneath the inferior rectus at an angle of about 52 degrees. After crossing the inferior rectus, it curves around the eyeball, making a large arc of scleral contact, and passes beneath the lower border of the lateral rectus to become inserted in the posterior lateral quadrant of the eyeball. The line of insertion is oblique and for the most part is below the horizontal meridian of the globe, with which it makes an angle varying from 15 degrees to 20 degrees.

In a comparison based on 100 specimens the anterior tip of the insertion of the superior oblique averaged 0.4 mm. anterior to the equator of the globe; whereas, the anterior tip of the inferior oblique insertion averaged 1.3 mm. posterior to the equator of the globe, a difference of about 2 mm.

Because the obliques are usually inserted in a different position on the globe, the oblique muscle planes do not coincide. However, most observers consider that, for practical purposes, the muscle planes should be regarded as the same, making an angle of about 52 degrees with the visual line, and that any mechanical difference in the plane of action is neutralized by the adjustment capacity of the central mechanism.

Undoubtedly, this is true in eyes in which slight deviation exists, but in cases of greater degree there is a question as to the validity of this assumption.

In a series of specimens studied,[2] measurements of the relation of the two obliques varied sufficiently to question such a supposition. In 129 adult human specimens, measurements were made of the relation of the planes of action of the two obliques to one another, and of their relation to the anteroposterior vertical plane of the eyeball.

The following data were found:

Average angle made by the oblique muscle planes with the anteroposterior vertical plane of the eye (based on the measurement of 112 specimens): Superior oblique 44.9 degrees (54 degrees usually accepted); Inferior oblique 45.5 degrees (52 degrees usually accepted)

Average measurement of 22 specimens showing greatest variation in the angles which the oblique muscle planes make with the anteroposterior vertical plane of the eye: Superior oblique 39.1 degrees; Inferior oblique 47.1 degrees

Average measurements of the 100 remaining specimens which showed less variation in the angle which the oblique muscle planes make with the anteroposterior vertical plane of the eye: Superior oblique 45.9 degrees; Inferior oblique 45 degrees

Although the average angle which the planes of action of the superior and inferior obliques made with the anteroposterior vertical plane of the eye was about 45 degrees in the second group, a study of individual specimens revealed in many instances an appreciable variation in the muscle plane of one oblique as compared with the other. They varied sufficiently from each other to raise a question as to a balanced action between the two muscles. It is apparent that other factors must act to compensate for the difference in the two planes of action in most instances of this type.

However, in higher degrees of variation, as found in some of the 22 specimens showing greatest variation, it is possible that such a condition may create a mechanical

obstacle to binocularity, and when associated with other etiologic factors, may be sufficient to produce a deviation.

The specimens showing greater variation could be divided into two main groups: (1) Some specimens showed a marked variation in the planes of action of the two obliques, (2) Other specimens showed almost similar angles with the anteroposterior vertical plane of the globe, but differed markedly because the oblique muscle planes were separated by several millimeters.

Measurements taken from individual specimens showed the greatest variation of the angle of the oblique muscle planes to be the following:

Measurements of the specimen showing the greatest separation of the oblique muscle planes: Superior oblique, 57 degrees; inferior oblique, 70 degrees.

Measurement of the specimen showing the greatest crossing angle of the two oblique muscle planes, superior oblique, 35 degrees; inferior oblique, 70 degrees.

Measurement of the specimen showing the greatest difference in the angle of the oblique muscle planes as compared with the anteroposterior vertical plane of the eyeball:

When the superior oblique shows the greatest angle: Superior oblique 64 degrees; inferior oblique 33 degrees.

When the inferior oblique shows the greater angle: Superior oblique 32 degrees; inferior oblique 57 degrees.

Measurements of the specimen showing the greatest angle of the oblique planes with the anteroposterior vertical plane of the eyeball: Superior oblique 67 degrees; inferior oblique 60 degrees.

Measurements of the specimen showing the smallest angles of the oblique planes with the anteroposterior vertical plane of the eyeball: Superior oblique 22 degrees; inferior oblique 34 degrees.

It seems to the author that a congenital maldevelopment in the planes of action of the oblique muscles is as logical an explanation as any for certain abnormal action of the vertical muscles. For example, take the case of the upshoot of the inferior oblique on adduction. That inherent lack of balance may exist between the opposing obliques is possible and can be explained on the following basis: The long course of human development has been associated with an increase in range of ocular movement and with an increase in accuracy over that range. During this process the superior oblique has surrendered some of that strength which, before the extension of its origin to the orbital apex, it used to balance with its antagonist. The resulting physiologic imbalance of the opposing obliques, coupled with an appreciable imbalance in the planes of action of the obliques in their relation to the anteroposterior vertical plane of the globe, may produce an upshoot or abnormal action of the inferior oblique, when the eye is adducted.

Also it is possible that certain cases of muscle imbalance, especially those seen in convergence and divergence anomalies can be due basically to a mechanical defect in the oblique muscle planes with the horizontal defect a secondary factor.

Such explanations are admittedly speculative, but there is some tangible basis for the assumption, which is based upon the above data. Such explanation may help to clarify some of the uncertain phenomena related to the complex etiology of the vertical defects.

REFERENCES

1. Brown, Harold W.: Strabismus: Ophthalmic Symposium, St. Louis, The C. V. Mosby Co., 1950, pp. 205-235.
2. Fink, Walter H.: Etiologic considerations of vertical muscle defects, Tr. Am. Ophth. Soc., 50:135-193, 1952.
3. Scobee, Richard G.: The fascia of the orbit; its anatomy and clinical significance, Am. J. Ophth., 31:1539-1553 (Dec.) 1948.
4. Whitnall, S. P.: Anatomy of the Human Orbit, ed. 2, London, Oxford University Press, 1932.

FIXATION DISPARITY

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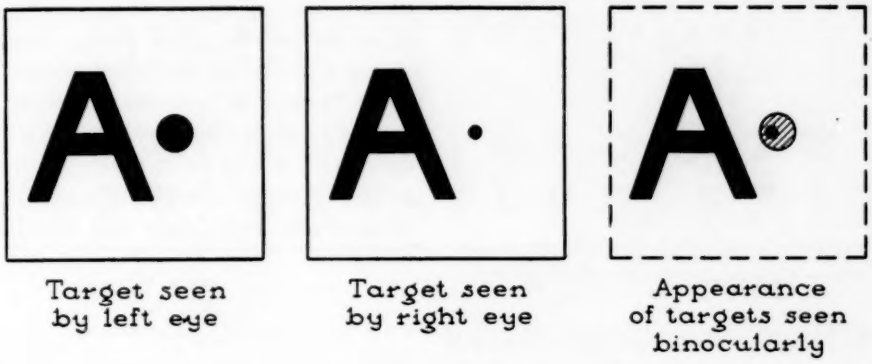


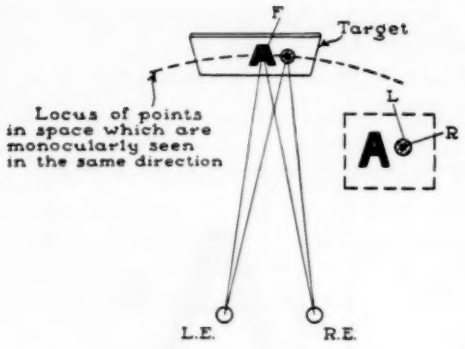
FIG. 1—Example of the type of targets that can be used to observe fixation disparity.

THE PHENOMENON designated fixation disparity has been known and observed for a long, long time. [1-4] It should be perfectly familiar to those of you who use haploscopic devices such as the stereoscope or amblyoscope. When targets or slides having some identical figures and also some dissimilar, or identifying marks are placed before the eyes, one observes that the images of the identical figures fuse readily, but the dissimilar marks will appear displaced with respect to each other if a heterophoria is present.

Figure 1 illustrates a suitable pair of such targets. The large letter *A* is identical and is accurately centered on each one. To the right of the letter on the same level and at the same distance is placed a disk—a large one to be seen by the left eye and a small one to be seen by the right eye.[1] When these two targets are observed in the haploscope (or by voluntary convergence), only a single letter *A* is seen. We say that the images of the two letters are fused. If no heterophoria (for the particular convergence of the eyes to the targets) is present, the small disk should appear in the center of the larger one. If there is a lateral phoria or muscle imbalance, the small disk will appear displaced within the large disk: displaced in the direction of the phoria. The direction and magnitude of this displacement of the small disk within the larger one can be changed by a change in the convergence angle of the arms of the haploscope, because then the muscle imbalance between the two eyes is *usually* changed. At some particular convergent position the two disks can be made to appear centered. Then for that convergence we can say there is no imbalance.

This displacement was described by Hofmann and Bielschowsky[2] as a *lag,* for they found that when the convergence positions of the two eyes were changed, this displacement was always in the direction opposite to the direction of convergence change. Ames and Gliddon[1] called the phenomenon a *retinal slip,* because the monocularly seen details appear to have slipped relative to the binocularly seen details. Neither of these terms adequately describes the displacement.

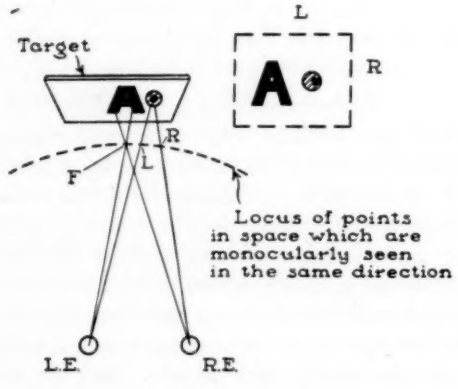
From the Mayo Clinic and the Mayo Foundation. The Mayo Foundation is a part of the Graduate School of the University of Minnesota.



ORTHOPHORIA

FIG. 2—In orthophoria where the monocularly seen images will appear in the same visual direction.

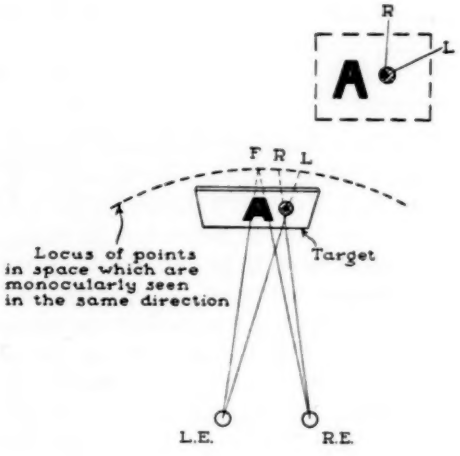
The basic fact about this phenomenon is that the subjective, visual directions of the monocularly seen details are different from the visual direction of the binocularly seen details. Now we know, of course, that within certain limits, images in the two eyes which are disparate—that is, do not fall on corresponding retinal elements—can be fused, that is, they are seen in one visual direction. This particular visual direction may correspond to the basic monocular direction of the retinal elements of one of the eyes or it may result from a compromise between the basic monocular directions of the retinal elements of the two eyes.



ESOPHORIA

FIG. 3—In esophoria where the monocularly seen images will appear in uncrossed disparity.

Now the phenomenon of fixation disparity must be due to the fact that the fused images, indeed, fall on disparate retinal elements. This disparity in turn must be due to the fact that the eyes are actually converging toward a point in front of, or behind, the target plane, depending on the direction of the lateral phoria. Any point "looked at" on the target is then imaged on disparate retinal elements. Thus the images of any point fixated on the target are disparate (fixation disparity). The monocularly seen details will actually appear displaced an amount equal to this disparity. Thus the phenomenon is an exhibition of a phoria while the fusion of similar images is maintained.



EXOPHORIA

FIG. 4—In exophoria where the monocularly seen images will appear in crossed disparity.

A full understanding of the phenomenon, of course, rests on our knowledge of the basic physiologic facts of binocular vision. We can better visualize the phenomenon from a study of figures 2, 3 and 4. Let us again use the target pattern just described, and again assume that by some arrangement the two disks are seen monocularly, the

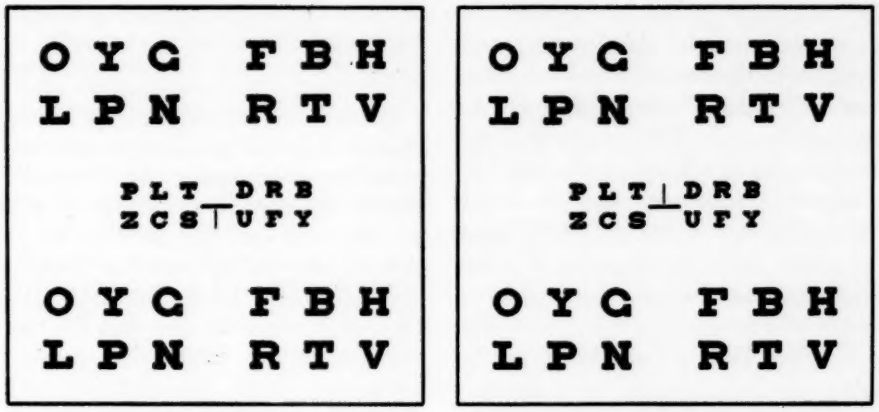


FIG. 5—Example of targets in which the nonius lines are used to observe fixation disparity.

small disk by the right eye, the large disk by the left eye. If the motor balance of the eyes is orthophoric for the particular convergence of the eyes (fig. 2), then the eyes will actually be pointing toward the plane of the target. Accordingly the horopter* lies close to the surface of the target. Hence the images of the two monocularly seen disks would appear centered, because the images of any point on the target surface would fall on corresponding retinal points and thus be seen in the same visual direction.

However, suppose there is esophoria (fig. 3). Then the eyes actually overconverge toward a point in front of the target. The images of the letter *A* then would be uncrossed disparate. The fusional processes, however, provide a single visual direction. But it will be clear that the image of the small disk will be seen to the right of the center of the large disk. Again, if the observer is exophoric (fig. 4), then the axes of the eyes actually underconverge or converge toward a point behind the target. The images of the fused details will then be crossed disparate. Here the small disk seen by the right eye will appear to the left of the center of the large disk. The overconvergence or underconvergence of the eyes cannot therefore be greater than the disparity corresponding to the maximal size of fusional areas (Panum's areas).

If there is a hyperphoria, the displacement of the small disk within the larger one will be in the vertical direction. This once more shows that the hyperphoria is manifested within the small limits of fusional areas, in spite of the fusion of the images of identical details on the two targets.

The two disks on the targets just described are not suitable for general use because their edges are too much alike. If the displacement is large, these two edges may be sufficient to provide a stimulus for fusion and as such would interfere with the test. A vernier or nonius type configuration has proved to be more suitable (fig. 5). These test details should not be used with pictures which provide stereoscopic depth, for the displacement of the two lines will vary with the different parts of the picture fixated.

*For a given point of fixation the horopter is that surface in space made up of points the images of any one of which monocularly seen by the two eyes appear in the *same* subjective visual direction. Therefore the images for that point are said to fall on corresponding retinal elements.

The magnitude of the displacement depends on the muscular imbalance between the eyes, on the fusional compulsion reflex, and on the type and pattern of details for fusion on the targets. Within certain limits, the more fusion detail, the smaller the fixation disparity. However, if these details are predominantly in the peripheral parts of the visual field, the disparity observed is larger, because Panum's fusional areas are larger there. Again, details for fusion near the fovea limit the magnitude of the disparity observed because here Panum's areas are smaller. However, even if the fusion details are near the fixation point (the test detail extrafoveal), a disparity can be shown to exist.[3,5] There are, of course, considerable individual differences. Usually if the compulsion-to-fusion reflex is poorly developed, the disparity will be larger and more unstable than if this reflex is well developed. The present point of view is that the magnitude of the disparity depends on the relative strength of the compulsion-to-fusion—the reflex to keep the eyes so directed that retinal images will fall as near as possible to corresponding retinal elements—and on the motor imbalance. The study of the phenomenon suggests that the strength of the compulsion to fusion itself varies with the extent of the disparity. This strength is least for small disparities but obtains a maximum when the disparities are such that diplopia is imminent. Thus the absolute magnitude of the disparity itself does not have precisely the same meaning regarding muscular imbalance as has the phoria measured by the Maddox rod.

The fixation disparity phenomenon is a valuable tool in the study of ocular motility, for it permits one to find the change of muscular imbalance while fusion, normal convergence and accommodation are maintained. The effect of prisms, or change in convergence, on the motor imbalance is easily studied. Similarly the effect of power lenses on motor imbalance through the accommodative-convergence synkinesis can be studied. Suppression can easily be detected.

Even with the simple type of targets described in the previous paragraphs, it is valuable to compare, for example, the phoria measured by the cover test with the Maddox rod and that convergence of the arms of the amblyoscope (or haploscope) for which the fixation disparity is eliminated, that is when the two dots are precisely centered or the nonius lines are aligned. Frequently these measurements will not agree in magnitude, though they usually will agree as to direction. Occasionally they will not agree even in direction. Also subjects will sometimes be found for whom no convergence can be found which will make the disparity zero, in spite of the fact that a phoria can be measured. We must insist that when both eyes are active and the images are fused, the muscular imbalance may be different from that which would be indicated by the usual methods of measurement where fusion is suspended.

For any particular target design, the actual angle of fixation disparity (the angular size of the displacement) can be measured if the nonius or vernier line on one target is made movable by a suitable screw and indicator[6-8]. With such a device the one line can be made to appear directly above the other. The actual displacement, namely, the fixation disparity, is thus measured. It is possible then to obtain a quantitative relationship between the fixation disparity and (1) forced convergence or divergence of the eyes by the use of prisms or by a change in angles of targets on the haploscope, and (2) the stimulus for a change in accommodation by placing spherical ophthalmic lenses before the eyes. The reader could profit from a study of the papers in which such relationships are discussed in considerable detail. Every orthoptist can readily make fixation disparity slides for the amblyoscope she uses, and then make use of this phenomenon in her work.

REFERENCES

1. Ames, Adelbert, Jr., and Gliddon, G. H.: Ocular measurements, Tr. Sect. Ophth., A.M.A., pp. 102-169 (June) 1928.
2. Hofmann, F. B., and Bielschowsky, A.: Ueber die der Willkür entzogenen Fusionsbewegungen der Augen, Arch. f. d. ges. Physiol., 80:1-40 (April) 1900.
3. Lau, Ernst: Neue Untersuchungen über das Tiefen- und Ebenensehen, Ztschr. f. Psychol. u. Physiol. d. Sinnesorg., Abt. 2, 53:1-35, 1921.
4. Lewin, Kurt, and Sakuma, Kanae: Die Sehrichtung monokularer und binokularer Objekte bei Bewegung und das Zustandekommen des Tiefeneffektes, Psychol. Forsch., 6: 298-357, 1925.
5. Ogle, K. N.: Researches in Binocular Vision, Philadelphia, W. B. Saunders Company, 1950.
6. Ogle, Kenneth N., Mussey, Frances, and Prangen, Avery deH.: Fixation disparity and the fusional processes in binocular single vision, Am. J. Ophth., 32:1069-1087 (Aug.) 1949.
7. Ogle, Kenneth N., and Prangen, Avery deH.: Further considerations of fixation disparity and the binocular fusional processes, Am. J. Ophth., 34:57-72 (May, Part II) 1951.
8. ———: Observations on vertical divergences and hyperphorias, A.M.A. Arch. Ophth., 49: 313-334 (March) 1953.

DIAGNOSIS AND TREATMENT OF ALTERNATING HETEROTROPIA

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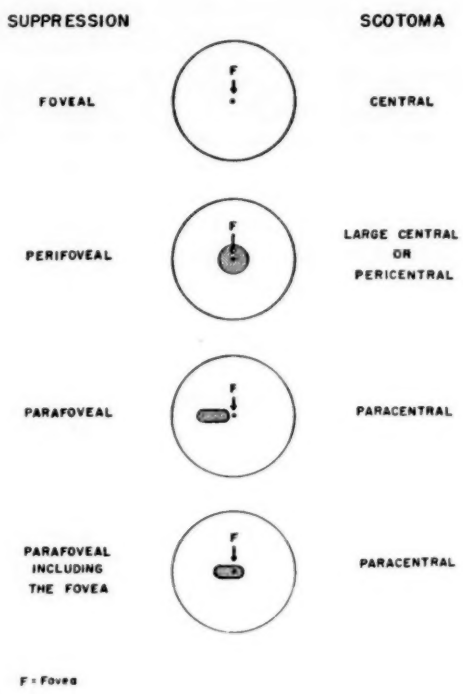
THIS STUDY of alternators was undertaken to provide a functional classification separating those cases in which fusion could most logically be expected from those in which a cosmetic result must probably be satisfactory, using the methods presently available to us. We shall also describe the methods we use in treating the various types of alternating heterotropia.

Alternating heterotropia is a manifest deviation of the eyes in which the patient is able to fixate, at will or unconsciously, with either eye and at the same time suppress, to some extent, the visual impulses from the nonfixing eye. The determining factor in making the diagnosis is the presence or absence of alternating suppression.

All patients who alternate fixation are certainly not alternators if one accepts our definition. The monocular deviator who is able to fix with either eye does not have the requisite alternating suppression. When he fixes with his dominant eye he suppresses the image before the nondominant one. He is not able to suppress the image before the dominant eye. If he is placed before the amblyoscope with an image before each fovea and the nondominant eye can be made to fix by oscillation or increasing the relative illumination of the targets before this eye, he will either fuse or have diplopia.

FIGURE 1



The adult who has had fusion all his life and suddenly develops a sixth nerve palsy is unable to suppress either eye, although he may be able to fix at will with either eye. He is constantly aware of diplopia. This patient is not an alternator.

Alternators usually have good visual acuity in each eye, but not always so. The alternator may prefer one eye for fixation most of the time. When this is so he may be mistaken for a monocular deviator.

The monocular deviator may become an alternator. This has occurred in our practice when a monocular deviator has been

This study was carried on in the Department of Ophthalmology, Oscar Johnson Institute, Washington University School of Medicine, St. Louis, and was supported in part by a research grant from the National Society for the Prevention of Blindness.

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Read before the annual meeting of the American Association of Orthoptic Technicians in Chicago, October 13, 1953.

patched until vision is equal and then the patch removed for a time before surgical realignment. Under these conditions, one will occasionally see a patient begin to alternate instead of returning to monocular suppression. We always try to keep the patch in place until surgery, patching the dominant eye each morning after the visual acuity is equal. This is done to prevent a return to monocular suppression, to prevent the patient from developing alternating suppression, i.e., becoming an alternator, and to prevent occlusion amblyopia.

We classify suppression areas in the eye as foveal (central), parafoveal (situated mainly to one side of the fovea, possibly, but not necessarily, including the fovea as a part of the suppression area), and perifoveal (an area almost equally surrounding the fovea and including it—this may be macular or extend any distance toward the periphery). This classification of suppression areas is useful in discussions of suppression and its terms are clear to most ophthalmologists as these terms have their counterparts in central, paracentral, and pericentral scotomas in visual field defects (fig. 1).

CLASSIFICATION

I. *"True" Alternators*

"True" alternators are those in whom it is impossible with the tests used to demonstrate diplopia. The "true" alternator has suppression which is complete as far as is demonstrable. When one eye fixes, the vision in the other is suppressed completely. The patient cannot be made aware of diplopia on the major amblyoscope at any setting with any targets. When diplopia cannot be demonstrated on the major amblyoscope, the patient is given red-green goggles and is seated not more than one meter away from an illuminated screen measuring three feet by three feet in an attempt to stimulate peripheral perception. The patient reports the color of the screen, and if at any time there is evidence that he sees both colors at the same time, simultaneous retinal perception is present. Another examination we use is the Lancaster red-green test in a darkened room. We have learned that this test will sometimes demonstrate simultaneous retinal perception when other tests fail.

The "true" alternator does not have, as far as we are able to determine, simultaneous retinal perception. We do not use the term "no correspondence" because an individual might have perception of visual impulses in each eye and still have no correspondence between the two visual impressions. The "true" alternator is unable to perceive any visual impulse coming from one eye while he is "seeing" with the other eye. He cannot possibly, of course, have any correspondence between the two images if he cannot see them at the same time, but that is beside the point. The thing that makes him a "true" alternator is the fact that he cannot perceive images from the two eyes at the same time. This is why we believe that "lack of simultaneous retinal perception" is better terminology for this condition than "no correspondence."

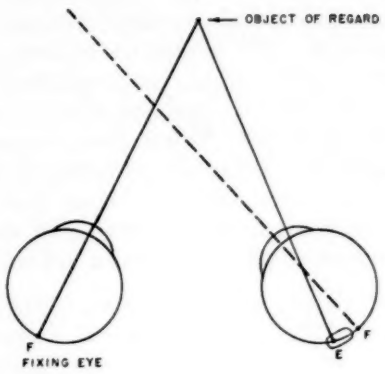
Treatment which may be used consists of (1) refraction to remove any accommodative element, to adjust the accommodation-convergence ratio, and give the best visual acuity possible; (2) surgery for realignment of the visual axes and (3) postoperative prisms, vertical or horizontal as indicated.

These patients are not amenable to active therapy from the standpoint of fusion training. Refraction in order to give them good vision and adjust the accommodation-convergence ratio is indicated. Surgical realignment of the visual axes should be done from both a therapeutic and cosmetic standpoint. Occasionally one will see a patient previously classified as a "true" alternator who, after surgical realignment of the visual axes, will begin to fuse or be converted to one of the other types of alternator.

The very deep suppression may break up of its own accord with time and surgical realignment, and when it does, orthoptics may be started. It is our opinion that surgery on these cases should be conservative and special care should be taken to avoid an overcorrection with a resultant exotropia. A narrow angle esotropia, well covered by the angle kappa, is frequently a desirable result surgically. Optical alignment of the visual axes can then be attained by horizontally based prisms incorporated into the lenses or prescribed as "clip-ons."

We are inclined to believe that these "true" alternators are similar to the subsequently discussed types, but that the suppression has progressed further. They are not necessarily hopeless, but they are not orthoptic material and must be given considerable time and careful watching.

FIGURE 2



F = Fovea

E = Extrafoveal area on which the image of regard falls when the other eye is fixing.

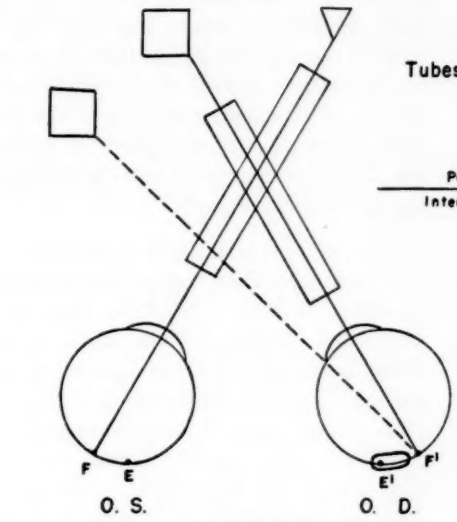
II. *Alternators with Abnormal Retinal Correspondence*

The alternator with ARC has alternating suppression, but he has a paracentral area of suppression, not including the fovea. That is, his area of suppression in the nonfixing eye extends as a horizontal bar from the extrafoveal point on the retina which receives the image of the object of regard of the fixing eye up to but not including the fovea (fig. 2).

This suppression area may be demonstrated by the use of a major amblyoscope with first grade targets, i.e., dissimilar targets which are agreeably superimposable. The machine is set at the patient's objective angle determined by "flashing", i.e., turning the lights on in the tubes alternately and changing the setting of the machine until his eyes no longer move when each fixes alternately. At this position the fixation spot is directly in front of the fovea of each eye. Let us presume that the targets are a box in front of the right eye and a top in front of the left eye. A patient who had normal correspondence and no suppression would see the top in the box when the tubes were lighted at the same time.

The alternator with ARC would see the box and top side by side with a space between them. If he has esotropia, he will have crossed diplopia at his objective angle. With exotropia the suppression area would, of course, be on the other side of the fovea and the patient would have uncrossed diplopia at his objective angle. In our experience ARC with exotropia is much less common than with esotropia. For purposes of discussion, we shall presume that the subject has esotropia and the left eye is the fixing eye; but it must be kept in mind that if the right eye takes up fixation, a suppression area similar to that which we shall discuss is found in the left eye. For simplicity, we have pictured only one eye fixing (fig. 3).

FIGURE 3



ALTERNATOR WITH ARC
Esotropia – Left eye fixing
Tubes placed at patient's objective angle
Foveal Stimulation each eye

Patient's Interpretation →

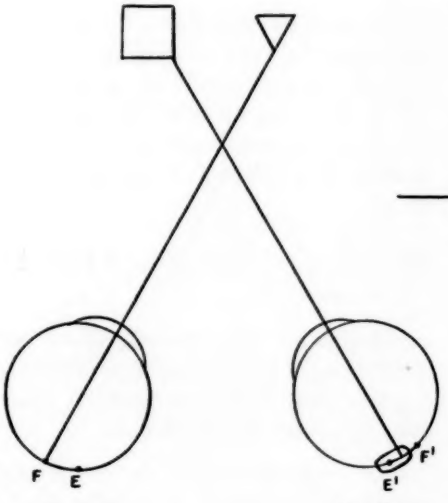
CROSSED DIPLOPIA AT PATIENT'S OBJECTIVE ANGLE.

[IN EXOTROPIA THERE WOULD BE UNCROSSED DIPLOPIA]

1. – – – – – – – LINE OF PROJECTION OF F'
2. F' AND E ARE CORRESPONDING POINTS.
3. CIRCLED AREA IN O.D. INCLUDING E' IS SUPPRESSED AREA.

With normal retinal correspondence patient would see ▽

FIGURE 4



ALTERNATOR WITH ARC
Esotropia - Left eye fixing.

Patient's Interpretation →

If the patient switched fixation to right eye with tubes in this position he would see only the box.

THE BOX IS NOW WITHIN THE SUPPRESSION AREA

If at this point the tube in front of the right eye is brought laterally (divergence) to attempt to superimpose the images, the box immediately falls into the suppression area and its image is lost to view. The patient will have alternate suppression rather than a subjective angle (fig. 4).

If the tube in front of the right eye were elevated so that the image fell on the retina below the area of suppression and the tube were brought laterally to an area below E′, the images would be seen as in figure 5.

The box can be brought beyond the suppression area (fig. 6).

In this manner we can circle and define the area of suppression. It is a horizontal bar extending from and including E′ to, but not including, F′. Any attempt to superimpose the two images will result in suppression of the box.

The alternator with ARC has simultaneous retinal perception in all areas outside of the suppression area.

Our routine in treating patients with this type of alternating heterotropia includes the following:

1. Refraction, to adjust the accommodation-convergence ratio, to remove the accommodative factor, and to give the best visual acuity possible.

2. Patching, combined with orthoptics to help break up the ARC as well as the suppression.

3. Preoperative orthoptics, to break up suppression and establish NRC.

4. Surgery, for realignment of the visual axes after NRC is established.

5. Postoperative orthoptics, to maintain NRC and build fusional amplitudes.

6. Postoperative prisms (either horizontal or vertical as indicated) to align visual axes optically after surgery if a small deviation still exists.

Patching tends to break up suppression, as it removes images completely from one eye and thus removes the need for suppression. It also eliminates the need for abnormal correspondence, as there is no need for projecting images abnormally to allow fusion. Either eye may be patched, but daily alternation of the patch or patching the dominant eye each morning probably is the preferable method.

Preoperative orthoptics to establish normal correspondence is much more valuable in the alternator with ARC than in the monocular deviator with ARC. Surgery alone is enough to break up the ARC in most monocular deviators and good surgical realignment is usually successful if combined with postoperative orthoptics. The alternator with ARC maintains his ARC at a new angle after surgery in many cases, and apparently surgical realignment is not enough to break it up. The job is made no easier by surgery, and if the new angle is very small it may be next to impossible, technically, to work with it. We use the Walraven technic* in working with these alternators to break up the ARC. Usually six to ten sessions are adequate to establish normal correspondence.

We have no explanation for the apparent preference for ARC in these patients. Theoretically surgery should be as effective an aid in breaking up ARC in alternators as it is in monocular deviators, but apparently, at least in our hands, it has not proven to be so.

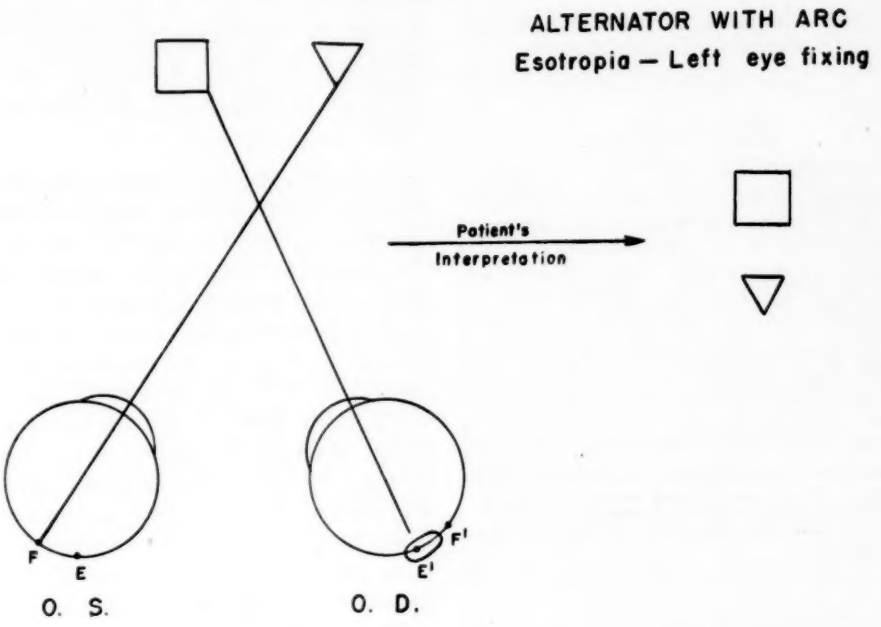
III. *Alternators with Normal Retinal Correspondence*

Alternators with normal retinal correspondence are the patients most likely to get a good result from treatment. A good result, of course, means fusion.

The suppression area in the nonfixing eye also is parafoveal. It includes the fovea

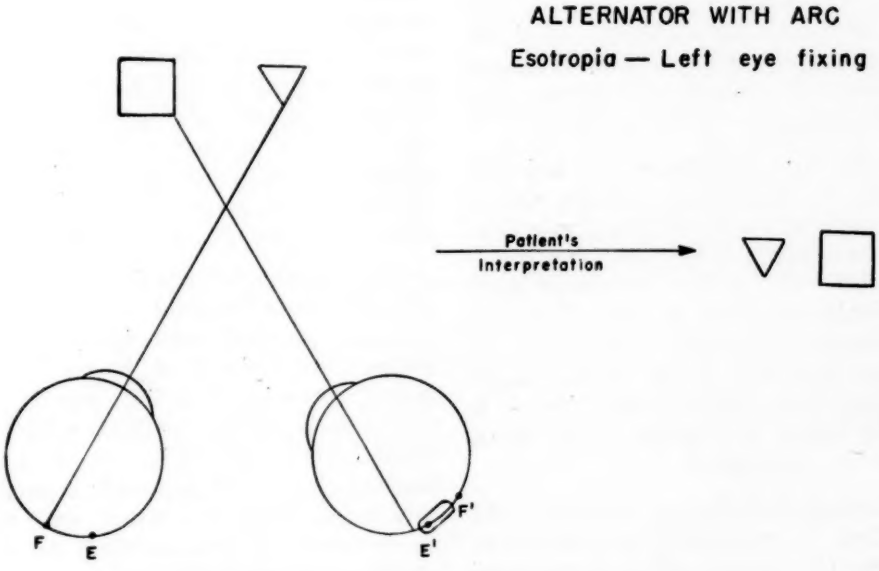
* Personal communication.

FIGURE 5



VERTICAL PRISM NOW REMOVES THE BOX FROM THE SUPPRESSION AREA

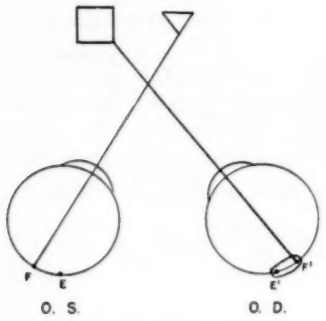
FIGURE 6



THE BOX IS NOW BEYOND THE SUPPRESSION AREA.

FIGURE 7

ALTERNATOR WITH NORMAL RETINAL CORRESPONDENCE
Esotropia - Left eye fixing



AT THE PATIENT'S OBJECTIVE ANGLE HE SUPPRESSES THE IMAGE IN FRONT OF THE NON-FIXING FOVEA. IF THE IMAGE IS MOVED VERTICALLY AND THEN LATERALLY, THE PATIENT CAN SEE BOTH IMAGES AND HIS SUPPRESSION AREA MAY BE MAPPED

and a horizontal bar extending out to include the extrafoveal area where the image from the object of regard of the fixing eye falls (fig. 7).

Simultaneous retinal perception can be demonstrated outside this area of suppression. In some patients the suppression area may be demonstrated only by foveal targets. It may extend toward the periphery, but it is not so complete that simultaneous retinal perception is impossible.

Frequently, with adequate stimulation by the use of unequal light intensity, oscillation, or flickering, superimposition and fusion are possible at the objective angle of deviation. The patient may have small amplitudes away from the angle. When fusion breaks in divergence, the patient suppresses alternately as one of the images is within his suppression area, but he is usually aware of diplopia when fusion breaks in convergence.

Treatment includes (1) refraction, (2) patching, (3) preoperative orthoptics, (4) surgery, (5) postoperative orthoptics and (6) horizontal or vertical prisms, if necessary. If a vertical component is present, the prescription of vertical prism frequently disrupts the established area of suppression sufficiently to encourage diplopia and may result in or aid in the development of fusion.

It is our practice to patch the eyes of those patients alternately preoperatively, or to patch the dominant eye each morning if dominance is expressed, and attempt to get fusion at the angle and work on amplitude training before surgery. In other words, we attempt to develop good fusion before surgery. We do not delay surgery long for this purpose, however, as these patients are easily amenable to postoperative fusion training, and with good surgical alignment they can aid their own cause postoperatively by using the eyes without a patch.

DISCUSSION

Those patients who cannot superimpose or fuse at their angle may have simultaneous retinal perception at some setting on the machine. At times it may be difficult to classify the patient definitely according to his correspondence. These patients should have patching, orthoptics to break up suppression, surgical realignment, and other treatment as indicated. Sometimes a satisfactory result is very difficult to achieve. Favorable changes after surgical realignment are seen in some patients after the passage of considerable time. Fusion may develop several years after surgery, even when active therapy has been abandoned.

Little mention is made in this paper of refractive error; however, we assume an adequate cycloplegic refraction is done in all cases. Esotropias should have a full hyperopic correction or as little myopic correction as is possible consistent with best visual acuity. Exotropias are refracted similarly, but a full hyperopic correction may not be given, or a myopic error may be overcorrected. Postoperatively the glasses prescribed are manipulated as may seem indicated, always, however, giving the full

cylinder and never prescribing lenses which cause any reduction in visual acuity. All patients are refracted two or three months after surgery to pick up any change in refractive error occasioned by the surgery, sooner if any manipulation of the prescription to aid fusion is contemplated.

Patching is usually done alternately, using an adhesive patch to occlude the eye completely. If any preference is expressed, the dominant eye is patched. The mother is told to patch the eye which is straight each morning when she changes the patch.

Postoperatively, if the surgical realignment is not perfect and a few prism diopters of deviation remain, we use "clip-on" prisms over the patient's glasses to compensate for this, and may later incorporate this into the lenses.

Alternators of middle age or over must usually be satisfied with a cosmetic result. Reasonably extensive therapy by patching is usually valueless and financial considerations, if the patient is the "breadwinner," or cosmetic reasons, if in college, frequently make patching and other therapy, which in this age group would necessarily be prolonged, unwise. A residual esotropia, well hidden by the angle kappa, is the most practical result to attempt here.

Developing fusion in alternators is, as a general rule, much more difficult than in monocular deviators; and for practical purposes the more difficult cases must be cooperative, live close by so that they may be seen often and return as often as necessary for therapy, and have sufficient funds for extensive orthoptics. We are frequently well satisfied with good vision in each eye and a good cosmetic result. From a practical standpoint these people have a good adjustment to their deviation and are only slightly inconvenienced by it.

DISCUSSION

JULIA E. LANCASTER, San Francisco, Calif.: Nothing but compliments can be extended to Lieutenant Stickle and Mrs. Laughlin for developing this analysis of one of the most knotty of orthoptic problems. Anything which helps us to classify orthoptic patients is valuable for both treatment and prognosis. Once we know what is preventing the patient's development of binocularity, we can plan efficient treatment without fumbling and waste of time. Moreover, the technician who can make reliable recommendations about prognosis is a jewel both to the ophthalmologists and to parents.

I think every technician would agree that the degree of difficulty in arousing binocular awareness is a close guide to the probable difficulty of eventually establishing fusion. As presented by Mrs. Laughlin, it is obvious that every possible skill and technic must be used to try to stop alternation. The less the skill of the technician the more likely will she be to conclude that it cannot be done. Before classifying a patient as a "true" or "truly stubborn" alternator be sure that no technic has been neglected which might lead to a more favorable classification. The only danger lies in making a diagnosis which postpones orthoptic treatment beyond the age of best learning. If on an early visit the patient is classified wrongly as unsuitable for orthoptic training, then he will not get the fusion experience on the amblyoscope which might be of more value to him while young than later in life.

It is important to balance the value of orthoptic training during the years of easy learning against the tedium of futile lessons for a patient whose chances of achieving binocular visual habits are remote.

DIFFERENTIAL DIAGNOSTIC CHARACTERISTICS OF INTERMITTENT EXOTROPIA AND TRUE EXOPHORIA

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THE MANAGEMENT of intermittent exotropia is one of the most confusing problems in the field of strabismus. Much of the confusion results from the lack of a clear definition of intermittent exotropia and failure to clearly differentiate it from true exophoria. It is the purpose of this paper to differentiate more clearly these two entities, which perhaps have a common etiologic basis but manifest quite different sensory-motor anomalies in the adult group, and require entirely different management.

Intermittent exotropia is one of the most variable of the ocular sensory-motor anomalies. There is seemingly a coexistence of two conditions. At one moment the patient may be fusing with normal retinal correspondence and full sensory fusion with relatively no symptoms. In another instant, one eye may assume a position of divergence unknown to the patient, and profound sensory anomalies may be manifest. At a different fixation distance the same patient invariably may demonstrate only one of these two conditions. It is not surprising, therefore, that considerable confusion exists in using the terms "exophoria" and "intermittent exotropia." The term "intermittent exotropia" (Lancaster[9]) is descriptive, is the one preferred by the Strabismus Symposium[12], is used in Philip Knapp's[8] recent excellent discussion and is to be preferred to other terms such as facultative exotropia and divergence excess.

It is possible to have one anomaly (such as constant exotropia) for distant fixation distance with profound sensory anomalies of suppression and anomalous correspondence, and to have a completely different anomaly (such as exophoria) for near fixation distance with no sensory anomaly demonstrable. When two such different sensory-motor anomalies can exist, each for a different fixation distance, it is obviously difficult to include both situations, and convey what is meant, by use of a single defining term. Definitions of intermittent exotropia found in the literature almost always include the term "exophoria" and attempt to include all fixation distances within the definition. Since intermittent exotropia is neither a wholly latent deviation nor always a manifest one, it constitutes a peculiar entity in so far as definitions of heterophoria and heterotropia are concerned.

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INTERMITTENT EXOTROPIA

Intermittent exotropia may be described as that condition characterized by a change in the relative position of the visual axes from parallelism to that of divergence, occurring unaware to a naive patient, without change in accommodation or in the object of regard. Such a patient has intermittent exotropia for that fixation distance, regardless of whether the phenomenon occurs rarely, occasionally, or frequently. Such a definition implies that intermittent exotropia may be associated with another different ocular sensory-motor anomaly at another fixation distance. For any given fixation distance, if an eye ever "wanders out" without diplopia, that patient has intermittent exotropia for that fixation distance. It will be noted that the term "exophoria" is not used within the definition and is reserved for a specific entity to be differentiated from intermittent exotropia. This is in sharp distinction from previously established custom.

Other definitions of intermittent exotropia (divergence excess) follow:

A. That form of ocular muscle imbalance which exhibits exophoria more marked for distance than for near, with normal prism convergence and near point of convergence, an excessive ability to overcome diplopia by prisms base in.[2]

B. *Divergence Excess*: Exophoria marked for distance, less for near (relations for near may be nearly normal). Convergence near point normal or nearly so. Prism divergence large (in typical cases disproportionately so; i.e., it is greater than seven degrees plus the exophoria for distance). Prism convergence often normal and affected with facility.[4]

C. *Divergence Excess*

1. For distance there is exophoria of five to thirty prism diopters or more. The power of divergence, as measured by the prism base in, is greatly increased and may be as high as thirty-three prism diopters. It is a constant factor, unvarying throughout repeated examinations. Lateral movements of the eyes are unaffected. There may be intermittent crossed diplopia, which is usually superable. This does not vary in amount in the lateral field, but diminishes as the test object is approximated to the patient.

2. For near objects, muscular deviation is within normal limits, and the near point of convergence is normal.[3]

D. *Divergence Excess*: Marked exophoria for distance. Excessive prism divergence. Normal prism convergence. Normal muscle balance at 13 inches. Normal near point of convergence. Lateral movements of eye in and out, normal in extent and comitant. Diplopia, if present, not increasing right or left.[5]

Other authors have chosen to assign an amount by which the distance anomaly must exceed the near anomaly, which further confuses the picture of just what constitutes the characteristics by which a case of intermittent exotropia may be identified. It is proposed that the diagnosis of intermittent exotropia may rest or fall on the fact than an eye "walks out"* under a given set of conditions described above.

In the descriptive definition of intermittent exotropia, the following factors are of importance: (1) fixation distance, (2) character of object fixated, (3) attention status, and (4) naivete of patient. These factors require further explanation.

1. *Fixation Distance*

One may have intermittent exotropia for either distance or near fixation, or both. Intermittent exotropia may be said to exist for distance and not for near fixation if an eye rarely, occasionally, or frequently "wanders out" while fixing distant objects but never does so for fixation of near objects. Intermittent exotropia may be said to exist for both distance and near if an eye at times "wanders out" for both distance and near fixation. Distance (6 meters) and near (40 cm.), are two rather arbitrary fixation distances that are clinically useful delineations in the diagnosis of any strabismus situation that may be different for one or the other of the two fixation distances. This is merely the application of descriptive terms for each fixation distance. With respect to the prognosis and management of various strabismus situations, it has more than academic differentiation.

The importance of differentiating the type of anomaly found at distance fixation from that found at near fixation was utilized by Duane[4] in his classification of motor anomalies into those of divergence and convergence. The descriptive terms used here for the exact characteristics of the ocular sensory-motor anomaly for each of the two fixation distances is comparable to Duane's classification of anomalies at distance fixation (of divergence) and at near fixation (of convergence) but, as Knapp[8] points out, avoids the etiologic basis implied in these terms. Although the Duane classification is still widely used, and although it is difficult to alter custom, there is a growing trend to use the descriptive type of nomenclature relative to fixation at distance and near as to whether there is never a frank involuntary tropia (phoria), occasionally a frank tropia (intermittent tropia), or always a frank tropia (constant).

* A term used in a similar connection by Linksz.[10]

2. *Character of Fixation Object*

The type of fixation target used during testing conditions may also influence the relative ease by which the exact characteristics of the divergent deviation become manifest to the examiner. A muscle light at 6 meters more frequently elicits the deviation than letters of equal size, perhaps because of the difference in attention required to fixate each. A fixation object requiring attention is less apt to reveal the full extent and exact characteristics of the divergent deviation. Similarly, objects fixated at greater than 6 meters distance are more likely to reveal the true characteristics of the deviation. White[13] recommended 45 to 60 meters in testing cases of divergence excess. It is obvious that the three factors of fixation distance, character of fixated object, and attention status are in some ways interrelated.

3. *Attention Status*

Inattention in some way helps trigger the mechanism that causes an eye to diverge (wander out) in intermittent exotropia. Attention is the key to the fusion mechanism. Inattention to the object of regard lessens the stability of fusion. Helmholtz[7] recognized the psychological factors involved in attention and its relationship to the fusion mechanism. The relationship of contour and attention has been described by Fechner[6], and its especial importance with regard to the condition of intermittent exotropia has been recognized by many authors. There may be degrees—more or less—of attention given to the object of regard. The case histories of intermittent exotropia bring out the importance of the factor of attention in revealing that the divergent deviation becomes manifest especially when the patient is tired, ill or daydreaming. Attempts to establish the diagnosis of intermittent exotropia may be aided or hindered by the relative degree of attention during testing conditions. One may not note the intermittent divergent deviation if the patient's attention is held securely by the object of regard at the time of examination. Sometimes the deviation will not become manifest even with repeated cover tests. If the parents of a child patient give a history of an eye "wandering out" at times, which is not revealed by cautious initial examination, the diagnosis of intermittent exotropia is by no means adequately ruled out. Re-examination with varying states of inattention may reveal that the parents were correct in their observation. With divergent deviations, the parents are usually correct; with convergent deviations, the examiner is usually correct.

4. *Naivete of Patient*

The descriptive definition of intermittent exotropia stated that the diagnosis was established if an eye diverged rarely, occasionally, or frequently, without the awareness of the patient. Not all patients with intermittent exotropia remain naive. Some patients are taught or learn tricks of awareness of eye position, have diminished suppression by orthoptic treatment, or in some way become aware of the deviation when it becomes manifest. These are tricks —very useful to the patient—which enable him to "catch himself" when the eye begins to diverge. The exact nature of this mechanism remains to be elucidated. Such patients may still have the passive and possible active factors operating in their ocular sensory-motor anomaly of intermittent exotropia and the evaluation of the permanency of such learned tricks remains unclear.

In summary, then, intermittent exotropia is a condition in which an eye "wanders out" without the awareness of the patient, under certain conditions of relative inattention. If an eye "walks out on a patient unannounced" rarely, occasionally, or frequently, while fixation distance, accommodation, and object of regard remain unchanged, then intermittent exotropia may be said to exist for that fixation distance.

DIFFERENTIAL CHARACTERISTICS

That some important differences exist between the conditions of exophoria and intermittent exotropia have been recognized by many authors, although the two continue to be rather confused and ill-defined. The most marked confusion results when intermittent exotropia exists for one fixation distance and exophoria for another fixation distance. Duane[4] was cognizant of this fact when he offered explanatory notes, remarking that it was "sometimes difficult to diagnosticate" one condition from the other following the coexistence of divergence excess with either convergence insufficiency or convergence excess. Bruce[3] stated that in the differential diagnosis of divergence excess, "the chief condition to be ruled out is convergence insufficiency." Bruce recognized this important difference and, relative to the conflicting evaluation of treatment which invariably follows conflicting and ill-defined definitions, found that "the disputants are not on common ground. Selecting the most articulate representative in each camp, one discovers that both Dunnington and Berens and his collaborators agree as to the findings for distance in divergence excess but differ in their ideas as to what the findings for near vision should be." And further, "one must conclude that Berens and his collaborators were treating the convergence insufficiency. It appears, then, that pure divergence excess is not amenable to orthoptic training, but that in cases in which convergence is affected, the condition may be expected to yield to this measure." The important point to observe here is that Bruce was cognizant of the importance of differentiating between the phoria and the intermittent tropia relative to treatment evaluation; but in discussing Bielschowsky's famous case of divergence excess[13], he stated that even though Bielschowsky believed that divergence for distance may be the normal position of rest in certain persons, "nevertheless, if a patient has excessive divergence, he has divergence excess." It is because of such confusion that an attempt is made here to more clearly differentiate these conditions.

EXOPHORIA

Exophoria may be described as that condition characterized by a wholly latent divergent deviation, never manifest except by conscious awareness and always with diplopia, for a given object of regard at a stated fixation distance without change in accommodative state. Such a patient has exophoria for that clearly observed object of regard.

It is clinically useful to use the terms "exophoria for distance" and "exophoria for near" for uniformity of descriptive nomenclature and, for purposes of this paper, to point out more clearly the differences between true exophoria and intermittent exotropia. More strictly speaking, one may choose to define exophoria as a condition confined to the distant fixation distance and describe it as the latent deviation measured by the fusion free position when the eyes fixate a distant object in the primary position with accommodation relaxed. If one uses the term "exophoria for near," one implies that the eyes are accommodated for the near object of regard and the latent deviation is now measured by an appropriate determination of the fusion free position.

Comparison of the descriptive definition of exophoria and that for intermittent exotropia reveals that the main differential point is that of the presence or absence of diplopia when the divergent deviation becomes manifest; in other words, the presence or absence of suppression when the eye is divergent. The extreme importance of suppression as the differential point will become evident in accounting for most of the differences in characteristics of the two conditions. The significance of suppression in the management of intermittent exotropia has long been recognized and has recently been emphasized by Knapp[8].

Exophoria is simply a matter of divergent position of rest of the visual axes which is held latent by the function of convergence. There is an absence of the sensorial adaptation of hemiretinal suppression found in strabismus to eliminate the diplopia when the eyes are manifestly divergent. Axial suppression (foveal, macular) of varying degrees may be present as in any heterophoria, but there is an absence of the hemiretinal type of suppression so characteristic of heterotropia[7a]. The hemiretinal character of suppression is demonstrable in intermittent exotropia whenever the eyes are divergent. In true exophoria whether the deviation is held latent or "allowed" to become manifest, a hemiretinal type of suppression is never demonstrable.

"Voluntary" Divergence

Some patients with a large degree of exophoria may "let" one eye diverge, with constant diplopia when so doing. This is a conscious trick and physiologically it is nothing more than relaxation of the convergence used to keep the divergent deviation latent. This type of so-called "voluntary" divergence is voluntary only insofar as the patient "allows" the function of convergence to relax so that the visual axes may assume the divergent position of rest.

Function of Divergence

That so-called "voluntary" divergence is an artifact in nomenclature may be shown by measuring the characteristics of the convergent and divergent fusional amplitudes. In exophoria there are usually clear-cut determinations of blur, break, and recovery points, the magnitudes of which substantiate the fact that there is a divergent position of rest with convergent and divergent fusional amplitudes balanced around this point. If prism divergence is measured from the zero (parallel axes) fusion position, one must remember that the initial increase in base-in prisms merely measures the relaxation of convergence until the amount of base-in prisms equals the phoria or position of rest. In exophoria the function of divergence is not being called upon to operate except relative to the divergent position of rest, and as a matter of fact, such measurements are often found to be low, probably because the divergent function is not much called upon in patients with exophoria*. Dunnington[5] and Posner[11] found high values of "prism abduction" but may not have clearly differentiated between the true exophoria and intermittent exotropia in comparing such determinations.

To determine the characteristics of the function of divergence in intermittent exotropia by the prism divergence method is by no means easy. Clear-cut end points and repeatable findings are not found as is the case with exophoria. Bair[12] has noted this confusion and remarked that "prism convergence and divergence are difficult to measure at distance in most cases because of suppression and lack of fusion." If prism divergence is measured in patients with intermittent exotropia so that the eyes may be directly observed, the following characteristics of the determination may sometimes be noted. The visual axes assume a progressively more divergent position in response to increasing amounts of base-in prisms introduced before the eyes. With gradual increase in the amount of base-in prisms, a point is soon reached when the visual axes rapidly assume a widely divergent position (as in the manfest phase of intermittent exotropia), during which time there is usually an absence of diplopia. If diplopia is present, it is extremely transient. From this point on there exists the strabismic type of hemiretinal suppression, and further increase in base-in prisms moves the retinal images over an area of hemiretinal suppression. When sufficient base-in prism has been introduced so that the retinal image crosses the foveal line into the nonsuppressed hemiretina, then diplopia invariably results. This determination, however, is in no way to be confused

* Adler[1] has recently demonstrated experimentally that during divergence from the convergent position, the lateral rectus muscles are actively innervated.

with a measurement of the function of divergence. This proposed mechanism may account for the high, constant prism divergence measurement found in intermittent exotropia by some authors.

To recapitulate, then, the sensory-motor findings in exophoria are no more mysterious than those found in normal patients with good fusion (of whom approximately 80 per cent have some heterophoria) except that in larger degrees of exophoria, some patients have learned the trick of relaxing the convergence necessary to keep the divergent deviation latent (or through fatigue) with invariable persistent diplopia as long as, and whenever, the divergence becomes manifest.

In exophoria, as in other true heterophorias, there is always only normal retinal correspondence. True heterophoria is in fact characterized by the absence of the strabismic type of sensorial adaptation of hemiretinal suppression and the occasional presence of anomalous retinal correspondence.

Exophoria in adults (over 6 years of age) is usually nonprogressive in degree throughout life, although the management situation may become aggravated when the presbyopic age is reached. Intermittent exotropia, on the other hand, may be progressive throughout life in both degree and frequency of deviation.

Exophoria may cause symptoms. The symptoms of heterophoria are described well in text books. The symptoms of intermittent exotropia are less well defined and are minimal when compared with those of exophoria. When fusing with the visual axes parallel, patients with intermittent exotropia, curiously, are relatively asymptomatic. It is precisely the inability of the adult patient with true exophoria to develop similar suppression that is responsible for the possibility of profound symptoms consequent to overcoming the divergent position of rest with a continued convergence effort. In intermittent exotropia the chief complaint is that of cosmetics; namely, that of the eye "wandering out." It is the profound suppression mechanism in effect when the eye is divergent that renders cases of intermittent exotropia relatively asymptomatic.

It may serve some useful purpose to summarize the differential characteristics of exophoria and intermittent exotropia in tabular form (table I). For the sake of completeness, a few points relative to management of the two conditions have been added.

Although it is not within the scope of this presentation to discuss the pathogenesis of the conditions, or their differential management, a few general remarks in this connection seem indicated. It is believed that exophoria and intermittent exotropia have a common etiology and usually arise during infancy. If the hemiretinal type of profound suppression is not developed during the time when it is capable of developing (namely, before the age of 6 years), then true exophoria persists throughout adult life and becomes primarily a problem of a motor anomaly. If the hemiretinal type of suppression does develop, then intermittent exotropia results, which may become more progressive throughout life. The problem then becomes one of profound sensory change, as well as of continued motor change. It is probable that an active factor in the divergence mechanism plays a role. These views will be elaborated upon in a subsequent publication.

Since adult exophoria has no profound sensory adaptation to the anomalous position of rest, the management of symptomatic exophoria becomes simply a problem of motor balance. That is, position of rest (fusion free position) in relation to the fusional amplitude, especially the convergence reserve. That is why any treatment directed at even partially improving the motor balance is effective in eliminating symptoms due to exophoria.

TABLE I

SUMMARY OF DIFFERENTIAL DIAGNOSTIC CHARACTERISTICS OF EXOPHORIA AND INTERMITTENT EXOTROPIA*

EXOPHORIA	INTERMITTENT EXOTROPIA
1. May "let" eye diverge momentarily (aware)	1. Eye "wanders" out (unaware)
2. Diplopia invariable when eye divergent	2. No diplopia when eye divergent (in naive patient)
3. Chief complaint (if any): asthenopia	3. Chief complaint (if any): cosmetic defect of eye "wandering out"
4. When fusing may be symptomatic	4. When fusing usually asymptomatic
5. Hemiretinal suppression absent under all conditions	5. Hemiretinal type of suppression profound when divergent
6. Magnitude of divergent position of rest relatively stable throughout life	6. Magnitude of divergent position and character of anomaly may become more profound throughout life
7. Only normal retinal correspondence found under any conditions	7. Either normal or anomalous retinal correspondence may be present when divergent
8. Prism divergence measurements relatively reliable	8. Prism divergence measurements unreliable and confusing
9. Function of divergence relatively normal without active factors	9. Probable active neurogenic factor in divergence mechanism
10. Management primarily a motor problem a. Treatment: A little prism, orthoptics, surgery, may alleviate chief complaint	10. Management primarily a sensory problem plus motor problem a. Treatment: A little prism, orthoptics, surgery, usually unsatisfactory in alleviating chief complaint

*It may be noted that most of the differential points listed are directly related to the factor of suppression.

A. A little base-in prism incorporated in ophthalmic lenses relieves the burden of continued convergence.

B. A little orthoptic training to increase the convergence amplitude is effective by increasing the amount of reserve in a taxed convergence function, which is fortunately responsive to the will and to this type of management.

C. A little surgery to improve, but not necessarily correct, the divergent position of rest is likewise effective in alleviating symptoms.

The management of symptomatic exophoria is sharply contrasted with the management of intermittent exotropia. A little improvement in the motor aspect of the problem is usually sufficient to alleviate symptoms of exophoria; whereas in intermittent exotropia a little prism, a little orthoptic training or a little surgery, directed at the same improvement of the motor aspect of the problem, usually meets with failure in correcting the chief complaint of the cosmetic defect of the eye "wandering out." In the management of intermittent exotropia special attention must be given to the sensory aspects of the problem, especially that of suppression, as well as to more than partial correction of the motor position of the visual axes.

REFERENCES

1. Adler, Francis Heed: Pathologic physiology of strabismus, A.M.A. Arch. Ophth., 50:19-29 (July) 1953.

2. Berens, Conrad, Hardy, LeGrand, and Stark, Elizabeth: Divergence excess; its incidence, its correlation with refraction, and the value of orthoptic treatment, Tr. Am. Ophth. Soc., 27:263-275, 1929.

3. Bruce, G. M.:Ocular divergence; its physiology and pathology, Arch. Ophth., 13:639-660 (April) 1935.

4. Duane, Alexander: A new classification of the motor anomalies of the eye, The Prize Essay of the Alumni Association of the College of Physicians and Surgeons, New York, 1896, p. 94.
5. Dunnington, John H.: Divergence excess; its diagnosis and treatment, Arch. Ophth., 56:344, 1927.
6. Fechner (Quoted by Hering, E.): Spatial sense and movements of the eye, Radde translation, Am. Acad. Optom., 1942, p. 37.
7. Helmholtz, H.: Physiological optics, vol. 3, p. 493 (Southall translation).
7a. Jampolsky, Arthur: Hemiretinal characteristics of suppression in strabismus. (To be published.)
8. Knapp, Philip: Intermittent exotropia; evaluation of therapy, Am. Orth. J., 3:27-33, 1953.
9. Lancaster, Walter B.: Terminology in ocular motility and allied subjects, Am. J. Ophth., 26:122-132 (Feb.) 1943.
10. Linksz, Arthur: Physiology of the Eye, vol. 2, New York, Grune & Stratton, Inc., 1952.
11. Posner, Adolph: Divergence excess considered as an anomaly of the postural tonus of the muscular apparatus, Am. J. Ophth., 27:1136-1142 (Oct.) 1944.
12. Symposium: Intermittent Exotropia, Am. Orth. J., 2:5-24, 1952.
13. White, J. W.: (Discussion) Bielschowsky, Alfred: Divergence excess, Arch. Ophth., 12:156-166 (Aug.) 1934.

STEREOPSIS IN FUSION TRAINING

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NEW YORK, N. Y.

THE ADVENT of three dimensional motion pictures has stimulated not only the layman concerning the entertainment possibility of stereoscopic vision, but the ophthalmologist and the orthoptist as well. We as orthoptists can readily appreciate the grand aid to visual training which this process places within our reach.

Cinerama is not three dimensional but depends for its effect upon the stimulation of peripheral vision, while central vision is being utilized. Three cameras of the conventional type take pictures of the scene. Three projectors flash the picture on an especially enlarged screen. The middle projector flashes pictures on the middle screen —the projector to the right flashes pictures to the left, and the projector on the left flashes pictures to the right. The effect is a panoramic, not a stereoscopic, view. The observer is engulfed by the scene; and he feels he is part of it.

With three dimensional films two stereoscopic camera lenses are used. When there is an increase in the separation of the lenses, the image will create the illusion of being extremely close to the observer, or when the separation between the lenses is decreased, the image will appear to recede. The pictures flashed on the screen are not perfectly superimposed, since they are taken from two different angles. The observer views the picture through polaroid lenses, with the axis of polarization of one lens at right angles to that of the other lens. Each eye sees a separate picture and when there is stereoscopic vision present a binocular depth sensation occurs.

The goal of the orthoptist is to make it possible for the strabismic subject to utilize this phenomenon which can be appreciated only by those who have binocular vision.

Presented at the Annual Meeting of the American Association of Orthoptic Technicians, Oct. 13, 1953, Chicago, Ill.

Visual acuity of both eyes is not greatly superior to one, nor is the brightness of objects perceived more than slightly greater in binocular vision. Since the phenomenon of stereopis is the only way in which binocular vision demonstrates itself as better than monocular vision it is well to review the facts of spatial localization.

We can attribute visual localization in space not only to binocular vision but also to certain factors which are referred to as empirical or secondary spatial clues and which are usually monocular in origin. Though stereopsis provides a more vivid and accurate relative depth discrimination, a perfection of spatial localization results from an integration of empirical and stereoscopic stimuli.

What are these secondary factors of stereopsis? Mainly they are the psychologic conceptions obtained by experience and learned by the stimulation of all the sense organs. Some of the more important factors follow.

1. *Perspective*: Linear perspective is the apparent convergence of parallel lines as they recede into the distance. The farther away images of equal size are, the smaller the retinal images. The extremely young child may see that the smallest cat in a picture drawn to scale is the smallest and his older friend will know that the smallest cat is the farthest. Thus the size of a retinal image related to a known size provides an important estimation of the distance of objects.

2. *Highlights and shadows*: They provide important clues by virtue of their shapes, intensity and their relationship to other objects. As an example, when light comes from above, the lower part of a projection which receives the illumination appears raised, the upper part indented. This empirical concept may be utilized by the artist to create an illusion of depth.

3. *Overlay*: Near objects tend to overlap and hide the more distant objects.

4. *Aerial perspective*: The edges of distant objects are less clearly defined owing to atmospheric haze. In a clear dry atmosphere images appear very close when in reality they are very far away. In foggy weather or when flying in mountain terrain, objects may appear farther away than they actually are.

5. *Height*: An object seen above others is viewed as farther away.

6. *Parallax*: This is the depth impression created by the displacement of unequally distant objects in motion, regardless of whether the objects move in respect to the observer, or the head and eyes of the observer move. This is a most important secondary clue to space localization. It is a highly developed response, and the precision of depth estimation thus attained is comparable to that of stereoscopic acuity. In motion parallax the relative motion must be of sufficient extent, velocity, and duration. Near objects are apparently retarded or with respect to the principal object of attention move to the side opposite the observer. The object of attention seems to be at rest, farther objects seem to participate in the motion unless the velocity is extremely excessive. In distance fixation, far objects seem to be at rest, less far objects somewhat retarded and near objects greatly retarded. In stereoscopic perception of moving objects the relative motion is a contributing factor and is more precise for an approaching than for a receding object. Motion parallax occurs not only in monocular vision but also in binocular vision as well. In the uniocular crescent of the peripheral field, depth localization depends entirely upon the factor of motion parallax; this is of much practical importance to the automobile driver or the landing pilot. The combination of these factors is responsible for the excessive stereopsis of stereo motion pictures.

What is the essential difference between stereopsis and depth localization attained through the afore-mentioned empirical fac-

tors? Binocular clues for stereopsis depend upon normal retinal correspondence, stability of fusion, and absence of suppression of any marked degree. Stereopsis depends upon the stimulation of horizontally disparate retinal elements. In the case of an exclusively vertical disparity there is no essential change in visual impressions. However, in the case of a horizontal disparity of simultaneously stimulated retinal elements, a new quality of sensation is added—a bulging out toward the observer or away from him. If the horizontal disparity exceeds the fusional area referred to as Panum's area, double vision occurs.

In near vision a far object stimulates disparate retinal elements which compared to the corresponding retinal elements are located nasalward. A nasal disparity creates the subjective impression of "farther away." The distance becomes increasingly farther the greater the nasal disparity. On the other hand, a near object stimulates temporally disparate retinal elements which lie temporalward to the corresponding retinal elements. A temporal disparity creates the subjective impression of "nearer to," and the impression of nearness is increasingly greater the greater the temporal disparity.

The stereoscope reveals the essential difference between the monocular and the binocular perception of depth. By means of suitable targets, disparities of the images of the two eyes can be introduced which are almost entirely free of secondary clues. A sense of depth will be perceived which is not evident when the image is viewed by one eye alone. We seem actually to see the empty space between objects. Stereopsis is therefore a specific response or sensation directly arising from physiologic stimuli. The empirical factors are psychologic, result from past experience and by their very nature are equivocal. Beyond 650 meters or a little less than a quarter of a mile, depth localization becomes entirely dependent upon these equivocal secondary factors, which accounts for erroneous judgment in distant space.

Convergence plays an essential role in the binocular judgment of absolute distance. On the other hand, in monocular vision only large sudden increases in accommodation are noticed or evaluated. In using the stereoscope, one of the first sensations of stereopsis which may be appreciated occurs when images are fused where the stereogram presents a gradually decreasing or increasing horizontal disparity.

What is the nature of binocular vision of those subjects whose eyes have a motor anomaly or disharmonious position? The study of this disturbed relationship has attracted the interest of the practicing ophthalmologist and the orthoptist as well as the theoretical scientist. First, this disturbance gives rise to double images of different sharpness; a sharp image of the object of attention, that formulated by the fovea of the fixating or dominant eye, and a less sharp image by the simultaneously stimulated eccentric area in the deviated eye. In the absence of binocular images on corresponding retinal points, the establishment of fusional areas is lacking and the basis for stereoscopic vision is no longer present. There occurs an inhibition of the extrafoveal image of the object of attention in the deviated eye, as well as the foveal area in the deviated eye. This phenomenon of inhibition is present not only in persistent normal correspondence but also in abnormal correspondence. When a new anomalous unity of the visual directions of the two eyes occurs, it may be restricted to a certain part of the visual field. The normal correspondence is displaced and under certain conditions it can reappear, possibly by changing the angle of deviation as by surgery or by simultaneous stimulation of both foveas. An attempt may be made to stimulate bifoveal fixation, while disparate peripheral retinal elements are stimulated simultaneously, creating a gross stereoscopic response.

It has been demonstrated that spatial distortions may be introduced through changes in the relative image size by means of certain lenses. People with high stereoscopic acuity may appreciate differences in the relative image size of less than a quarter of a diopter. The space eikonometer for aniseikonia examinations utilizes this factor.

When the eyes turn to observe a near object in asymmetric convergence, difference in the sizes of the two images will be expected because the distance of the object from one eye will be greater than from the other. The difference will be greater the nearer the object is to the eyes, and the greater the degree of lateral turning. If one observes, for example, a coin in near vision with asymmetric convergence and the eyes are allowed to overconverge or underconverge slightly so that the object is seen in double vision, the image seen by the nearer eye appears larger. Under normal conditions monocular images of slightly different form and size are somehow combined into a single binocular image.

Ames in 1931 found that when the arms of the haploscope were rotated in asymmetrical convergence there was an apparent change in the size of the targets. The apparent change in relative size was in the direction which would have corrected the difference in the sizes of the images had the eyes been observing under actual conditions with the same degree of asymmetric convergence. This apparent change in size with lateral gaze becomes greater as the targets are placed nearer to the eye, and is of such slight degree that it is not easily observed at six meters.

In summary we may note:

1. Stereopsis requires simultaneous vision in both eyes and bifoveal fixation.

2. Stereopsis occurs over the entire binocular field and is not limited to the area of fixation. Stereopsis is usually measured in or near the macula, because there we find the keenest discrimination of depth.

3. Stereoscopic acuity is usually related to the monocular visual acuity of the two eyes, though there is some variation in high degrees of stereopsis.

4. Stereopsis may be entirely absent, though there be a high degree of visual acuity in each eye.

5. Stereoscopic acuity will depend upon the same factors which affect visual acuity, such as illumination.

6. Stereoscopic acuity depends upon the duration of the stimuli.

7. There is a correlation between empirical clues and binocular stereoscopic acuity.

8. The disparity of images in the two eyes provides the stimulus for stereoscopic sensation as well as fusional movements.

The phenomenon of stereopsis continues to be a challenge for further study. What has been uncovered is merely an introduction, not a satisfactory theory of visual space. Further developments will require the efforts of those leaders who are investigating sensory relationships.

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Editorials

ANNUAL MEETING OF THE AMERICAN ASSOCIATION OF ORTHOPTIC TECHNICIANS

The twelfth annual meeting of the American Association of Orthoptic Technicians was held at the Palmer House in Chicago on October 11, 12 and 13, 1953.

The first presentation of the Walter B. Lancaster Award was made to Miss Julia E. Lancaster for her outstanding contribution in the field of Orthoptics.

Winners of the 1953 slide contest were announced. First prize was awarded to Miss Mary Virginia Stallworth of Birmingham, Alabama; second prize to Miss Elizabeth Stark of New York, and third prize to Miss Dorothy Bair of Washington, D. C.

A group Insurance Plan was approved by the American Orthoptic Council. However, it was not fully subscribed and will be offered again in 1954.

An attraction among the Scientific Exhibits was the exhibit organized by Miss Mary Kramer, on "Accommodative Esotropia," which correlated with the symposium.

Another exhibit on "Occlusion" was given this year in New York by invitation of the National Association for the Prevention of Blindness at their convention. Co-chairmen for this exhibit were Miss Geraldine Wilson and Miss Evelyn Taylor.

Following the general meeting the various sections gathered and elected the chairmen and locations for the 1954 Regional Meetings. The results are as follows:

1. Western Section at Portland, Ore.
   Ruth Wahlgren, chairman
2. Midwestern Section at Rochester, Minn.
   Mary Cronin, chairman
3. Eastern Section at Washington, D. C.
   Maria Borzillo, chairman
4. Southern Section at Florence, S. C.
   Marguerite J. Worsham, chairman

BENEVOLENT UNCLE!

FEW orthoptic technicians realize the contribution made by one professional organization to the growth and enlightenment of orthoptics.

When the American Orthoptic Council was formed in 1938, three sponsoring organizations collaborated to make the dreams of Miss Elizabeth Stark and Dr. LeGrand Hardy a reality. They were the Section on Ophthalmology of the American Medical Association, the American Ophthalmological Society, and the American Academy of Ophthalmology and Otolaryngology, and soon thereafter these were joined by the Section on Ophthalmology of the American College of Surgeons. The continuing encouragement and support of these organizations have made possible the greatly expanded activities of the American Orthoptic Council, the formation and growth of the American Association of Orthoptic Technicians, and the evaluation and greater appreciation of orthoptics in the large field of ophthalmology.

Because of its focal position, one of these organizations, with its secretary-treasurer, has been particularly helpful. The American Academy of Ophthalmology and Otolaryngology and Dr. William L. Benedict have given inspiration and implementation to many of the most valuable activities of the American Orthoptic Council and the American Association of Orthoptic Technicians.

The Academy has made available a meeting room and announced in its general program the annual "symposium on orthoptics," jointly sponsored by the American Orthoptic Council and the Association of Orthoptic Technicians. This program has, during the past several years, occupied a position of increasing importance in the discussions of strabismus.

The Academy has been particularly generous to the individual certified orthoptic technician. For several years it has allowed them to register without fee and attend all general meetings and exhibits at the annual Academy meeting. Further, the American Association of Orthoptic Technicians has been allowed each year to present an exhibit among the scientific exhibits. During the past three years the exhibit has been especially valuable since it has been closely correlated with the subject matter of the annual symposium.

Two years ago, the Academy, with the cooperation of Dr. A. D. Ruedemann, initiated instruction courses solely for orthoptic technicians. These courses (twelve in the first year and fifteen last year), covering the broad subjects of binocular vision and ocular motility, have been oversubscribed each year and give evidence of the orthoptic technicians' desire for advanced study. Both the physical arrangement and the subscription for these instruction courses is handled by the Academy.

The latest proof of the Academy's substantial interest in ocular motility and orthoptics is the American Orthoptic Journal, the fourth issue of which is in your hands. Through the efforts of the late Dr. Richard Scobee, and with the full and continuing cooperation of the Academy and Dr. Benedict, the American Orthoptic Journal is growing and maturing with each annual publication. Now, not only does the orthoptic technician have an annual meeting, instruction courses, exhibit, and attendance privileges at the annual Academy meeting, but it also has a "printed voice," the American Orthoptic Journal—all through the cooperation and courtesy of the American Academy of Ophthalmology and Otolaryngology.

It is impossible for the individual technician to express her or his appreciation to the Academy for all of this, other than by a continuing growth in professional knowledge and continued adherence to the best principles of professional ethics and practice. "Uncle" Academy has been unusually good to us.

FRANK D. COSTENBADER, M.D.

REMEMBER SUPPRESSION!

IN A recent discussion of the standards that orthoptic technicians should meet, it was stressed that many of the basic concepts in our field are not sufficiently emphasized. It is imperative to stress upon the young student the importance of making a very thorough study of the sensorial aspects before embarking upon the luring course of research.

One of the important points brought up in the discussion which prompted this article was the fact that some ophthalmologists and even some orthoptic technicians tend to pass over the diagnosis and treatment of suppression.

Suppression can exist in various forms and degrees. It may be peripheral or central, deep or slight, monocular or alternating. A person must be trained to understand that even though the patient shows no peripheral suppression there may be central suppression present. The trainee should be made to realize the importance of making a correct evaluation of this sign.

The fundamental aim of orthoptic training is to eliminate suppression, both peripheral and central. Too often one is satisfied with eliminating peripheral suppression without regard to the more difficult task of eliminating central suppression. Knowledge of the degree and density of suppression is essential in making an accurate prognosis as to the type and duration of treatment. Therefore, one must first test for its presence and then treat it systematically.

The ultimate success of orthoptic training depends upon the careful study and observance of this simple basic principle.

NANCY M. CAPOBIANCO

REGIONAL MEETINGS

THE 1953 regional meetings were well attended and much interest was shown in programs prepared by the respective chairmen.

These mid-year meetings provided opportunities for the exchange of ideas which pertained especially to the practical phases of orthoptics. The clinical setting and freedom from congestion made it possible to include case studies and demonstrations as well as round table discussions. A complete report on the findings and recommendations of the ethics committee of the American Association of Orthoptic Technicians was read and discussed at each section meeting.

Many of the papers prepared for these meetings were published in the 1953 American Orthoptic Journal along with the Symposium papers from the previous annual meeting.

The attention given to home training was noteworthy. "Be it ever so simple," a little task (ranging from monocular fixation to physiological diplopia) performed daily can be a stepping stone toward better binocular coordination at the next office visit. An alert orthoptist knows when conditions are favorable for supervised homework and also possesses that "sixth sense" which tells one when any attempt at home exercises creates a greater problem than that which already exists.

Other speakers at the spring meetings were chosen to present subjects closely allied to orthoptics. Judging from the comments which ensued, orthoptists are anxious to learn more about treating the child as an individual rather than as a "pair of eyes." It may be easier to follow a set of proven principles automatically than it is to stop and weigh each problem; our technical training is invaluable in helping us to work quickly and accurately but the orthoptist must be a TECHNICIAN PLUS.

Certainly our regional meetings, together with the annual meetings, give us a well-rounded program for the year. They also provide a brief respite from office routine at a time when this is most welcome. Be sure to attend your regional meeting in 1954!

ELSIE H. LAUGHLIN
Vice-President, 1952-1953

THE AMERICAN ORTHOPTIC COUNCIL — 1954

The American Orthoptic Council is composed of three representatives each from the American Ophthalmological Society, the Section of Ophthalmology of the American Medical Association, the American Academy of Ophthalmology and Otolaryngology, and the American College of Surgeons. Four associate members are elected from the American Association of Orthoptic Technicians.

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Committee on Delta Gamma: Dr. LeGrand H. Hardy, representative.

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AAOT Exhibit for Academy and International Congress (1954)

To be appointed

Exhibit
National Society for the Prevention of Blindness

To be appointed

Abstract Department

The section on abstracts was made possible by the able assistance of Dr. Bruno Bagolini and Mrs. Shulamit Leibel and our abstract committee, consisting of:

Norma Glaser
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Jane Hall
Jean Swenson
Dixie Wehrheim

CLASSIFICATION

1. Amblyopia
2. Correspondence
3. Depth Perception
4. Dominance
5. Esotropia
6. Fusion
7. Instrument
8. Miotics
9. Miscellaneous
10. Nystagmus
11. Occlusion
12. Physiology
13. Reading Problems
14. Suppression
15. Technics
16. Treatment
17. Vertical Deviations
18. Visual Acuity

1-1

Jaffe, Norman S., and Brock, Fred W.: *Some phenomena associated with amblyopia,* Am. J. Ophth., 36:1075-1086 (Aug.) 1953.

The authors investigated the problem of whether there is foveal fixation in amblyopia. The procedure of their investigation involved the principle of "transferring" the awareness of the afterimages from one eye to the other. The amblyopic eye is occluded, a vertical bar of light is presented to the nonamblyopic eye for 20 seconds. The nonamblyopic eye is covered and the patient fixates with the amblyopic eye the central dot on a chart that is arranged much the same as a tangent screen. If both eyes are normal, the patient reports an afterimage running vertically through the dot. Where no correspondence exists there is no awareness of the afterimage in the exposed eye. If the "transfer" is reported running vertically through the dot then the nonexposed eye or amblyopic eye is fixating with the fovea. If the amblyopic eye uses an eccentric retinal area for fixation, the patient will report the "transferred" afterimage as being eccentric to the dot. Diagrams of steps in the procedure are presented.

The subjects consisted of 107 anisometric amblyopes, 98 strabismic amblyopes, 48 alternating strabismics without amblyopia, and 40 patients with correctible refractive errors. Specific results in typical cases are reported. Tables of results are also presented. The authors conclude that many patients with amblyopia have eccentric fixation.

Since fixation is eccentric in many amblyopes, "the depth of amblyopia is directly related to the eccentricity of fixation." The more eccentric the fixation, the deeper the amblyopia. It was also found that most of the patients who demonstrated an eccentric "transfer" also had a central scotoma.

The cases of 20 amblyopic patients, aged 6 years to 23 years, who had total occlusion therapy for a maximum period of three and a half months, are summarized.

The authors conclude that if central fixation exists, occlusion therapy is satisfactory. However, the vision of the amblyopic eye did not improve when there was an absolute central scotoma. It did improve when the scotoma was very small and the area of eccentric fixation variable.

1-2

Kasser, M. D., and Feldman, J. B.: *Amblyopia in adults, treatment of those engaged in the various industries,* Am. J. Ophth., 36:1443-1446 (Oct.) 1953.

The authors present a preliminary report on the treatment of amblyopia in adults. Most recognized methods, such as patching or atropine, are impractical for adults and cannot be used. For the treatment of amblyopia the telescopic amblyoscope has proven to be quite successful with children if the treatment was started early. In adults, treatment with the telescopic amblyoscope has been less successful due to irregularity in attendance and a tendency to stop treatment as soon as improvement was noticed. Progress rate depended upon the endeavor of the individual, even in similar cases with the same type of treatment.

2-1

Jaffe, Norman S.: *Anomalous projection; its incidence, factors in development, characteristics, tests, and treatment in* 146 *surgically treated strabismus cases,* Am. J. Ophth., 36:829-838 (June) 1953.

The author discusses the incidence of anomalous projection as reported by various investigators and compares the incidence in his own series of 146 strabismus cases. Factors in the development of anomalous projection are discussed, age of onset, the degree of squint, rapidity of onset, convenience to patient, and emotional environment.

He describes characteristics of anomalous projection, such as the angle of anomaly and the suppression areas. Five tests for anomalous projection are described and discussed including the afterimage presented to just one eye. The author states that when there is correspondence the afterimage is seen by the opposite eye. If it is not seen by the opposite eye there is no correspondence.

Both nonsurgical and surgical types of treatment are discussed, and the author presents a series of 48 adult strabismus patients with anomalous projection in which 57 per cent achieved correspondence postoperatively by surgery alone.

2-2

Rønne, Gerhard, and Rindziunski, Eva: *The diagnosis and clinical classification of anomalous correspondence,* Acta ophth., 31:321-345, 1953.

The tests which were used to determine correspondence in the subjects for this paper were: afterimage, haploscope, diplopia, parallel vertical afterimage, prism-rack afterimage, prism-rack Maddox rod, and afterimage at varying distances.

A series of cases are presented which illustrate each of the different classes of anomalous correspondence. The five classes, or stages, of anomalous correspondence are:

1. The earliest stage in which normal correspondence dominates. The patient has fusion on the haploscope, usually with stereopsis, suppression for diplopia test, and diplopia for Worth four light. The afterimages are usually normal, but the parallel negative afterimages have a tendency to separate.

2. On the haploscope there is macular suppression with small pictures, as a rule the patient has fusion with large pictures, but no stereopsis. With Worth four light there is alternating suppression, and the findings with Maddox rod are often normal, but sometimes the subjective is less than the objective angle. The afterimages are normal, but the negative crossed afterimages are sometimes anomalous or alternately suppressed. The parallel afterimages are usually anomalous.

3. On the haploscope instead of simultaneous perception there is frequently a "jump" in the subjective angle. Fusion is sometimes possible with large pictures. Worth four light is suppressed, and with the Maddox rod the subjective angle is less than the objective angle. Positive crossed afterimages are usually anomalous, negative usually showing alternate suppression or being anomalous.

4. At the haploscope simultaneous perception at a subjective angle not far from zero, and sometimes fusion within the subjective angle. There is suppression with Worth four lights, and a small subjective angle with Maddox rod. It is usual to find anomalous afterimages with a constant angle of anomaly, however sometimes the images can be brought together with the prism-rack test.

5. At the haploscope there is simultaneous perception with small pictures at a subjective angle near zero. Fusion sometimes is easy, and there may be some amplitude, but no stereopsis. There is some suppression with Worth lights, and with the Maddox rod there is harmonious or nearly harmonious anomaly. The afterimages are almost always anomalous with the constant angle of anomaly indentical, or nearly so, with the objective angle.

This classification is important for two reasons; to evaluate the prognosis, and to determine the therapeutic measures necessary. The first three stages have a better prognosis and indicate that the therapeutic measures need not be as prolonged or as radical as might be indicated by the later stages.

2-3

Rønne, Gerhard, and Rindziunski, Eva: *The pathogenesis of anomalous correspondence,* Acta ophth., 31:347-366, 1953.

This paper begins with a discussion of Traver's theory of anomalous correspondence, and a general acceptance of it as an explanation. Several cases are cited to reinforce the theory that anomalous correspondence is a process of adaptation.

The authors state that the most efficient way of combating anomalous correspondence is by use of the Walraven technic utilizing monocular diplopia. They found that in some cases which did not respond to treatment over a period of months they achieved almost immediate results with monocular diplopia.

The evolution of anomalous correspondence is, in the authors' words, " . . . perhaps mediated through hypothesized transverse couplings between the two layers of ganglion cells, separated by the white line of Gennari, characteristic of the striate area in the occipital cortex. Anomalous correspondence should consequently be regarded as an anomaly of perception, and the adjective, retinal, omitted."

The necessity of early diagnosis and treatment is emphasized by the authors. As is common in other acquired anomalies, it is easier to prevent than to cure, so the authors have emphasized prophylaxis from two angles; occlusion, and early correction of the squint by surgical, optical or prismatic means.

3-1

Miles, Paul W.: *Anomalous binocular depth perception due to unequal image brightness,* A.M.A. Arch. Ophth., 50:475-478 (Oct.) 1953.

Attention is called by the author to an anomaly of depth perception described by a monocular defect of unequal brightness of retinal images called anisopia. Anisodominance is also an anomaly of binocular vision and differs from aniseikonia or anisopia in that stereopsis is not required. It causes simple errors of relative distance of objects of any shape. If an image appears brighter to one eye than the other, the brighter one of the two objects appears nearer.

Facts contributing to anisodominance are (1) brightness and size clues to distance with relation to dominance; (2) illumination reduction in one eye causing loss of stereoscopic acuity; (3) greater brightness needed to recognize the images equally in the suppressing eye with normal visual acuity than with the dominant eye as in the mirror haploscope.

Further tests were demonstrated with the Lancaster red-green goggles, and white cylinders made from typing paper pasted on black cardboard and smoked glasses to be effectively used, substantiate the phenomenon.

4-1

Berner, George E. and Berner, Dorothy E.: *Relation of ocular dominance, handedness and the controlling eye in binocular vision,* A.M.A. Arch. Ophth., 50:603-608 (Nov.) 1953.

Crossed dominance has long been thought to be at the root of many reading and writing disabilities. The Berners come forth with an hypothesis based upon a study of more than 500 patients with and without reading and speech difficulties. A distinction is made between the dominant (the sighting eye) and the controlling eye (the eye that controls binocular perception). It is pointed out that they are not automatically the same. While the dominant eye is stable, the controlling eye can be changed. The Berners' investigation led them to believe that reading, writing, and speech difficulties arise when there is crossed control (right hand-left eye control). While many patients with crossed dominance have difficulties, it is noted that the circumstances which cause crossed dominance can also cause crossed control.

The examination routine, and analysis of the cases used are presented. Treatment is directed towards shifting the controlling eye to the side of hand dominance.

5-1

Nordlöw, W.: *Age distribution of the onset of esotropia,* Brit. J. Ophth., 37:593-600 (Oct.) 1953.

This statistical analysis is based on a series of 485 cases collected during a ten-year period. A comparison is made with previous studies made between 1899 and 1951, and consideration is given to possible sources of error in the methods employed, such as over-representation of the early age groups due to the amount of time elapsed before the patient's first examination.

The conclusion is drawn that esotropias develop more frequently during the first year of life than at any other time. This refutes the earlier idea that more esotropias develop between the ages of 2 and 4. A difference is found, however, between the onset of constant and intermittent esotropia. The older the child is at the onset of squint the more frequent is the occurrence of the intermittent squint.

6-1

Winkelman, J. E.: *Central and peripheral fusion,* A.M.A. Arch. Ophth., 50:179-183 (Aug.) 1953.

The author distinguishes between motor and sensory fusion and further defines sensory fusion as a perception and motor fusion as a reflex. The perception (sensory fusion) of identical images creates motor (reflex) action causing the identical images to fall on corresponding points of the retina. Burian has shown that identical peripheral stimuli give rise to fusional movements capable of breaking foveal fusion. The peripheral retina creates greater motor fusion than central vision where motor activity is less pronounced. Experiments were performed using retinal rivalry as a basis because it more nearly represented the sensation of squinters. From these experiments it was concluded that with exposure of images giving rise to peripheral retinal rivalry a fusional movement of the eyes was noted. This in the author's opinion is essentially a primitive reaction to light stimuli. It is not impossible that this reflex is present in squinters. These experiments and the findings of Burian, who proved that even patients with anomalous retinal correspondence may react to peripheral stimuli by fusional movements, could be explained as constituting the remnants of a probably congenital and unconditioned reflex to light.

7-1

Ellenberger, Carl: *Homemade apparatus for testing retinal correspondence,* Am. J. Ophth., 36-1121-1122 (Aug.) 1953.

This article explains how one may construct an apparatus to test retinal correspondence by using the afterimage as described by Tschermark. A picture of the apparatus and a wiring diagram are included. The sockets are placed in a wooden base so that the frosted bulbs are at right angles to each other forming the letter "L" backwards. The lights can be turned on alternately by a toggle switch.

The author describes his procedure and advises that the dominant eye be exposed to the horizontal bulb and the nondominant eye to the vertical bulb. Instead of having the patient draw a picture of the afterimage the author prefers a verbal report.

7-2

Fink, Walter H.: *Chart for testing near vision,* Tr. Am. Acad. Ophth., 57:-112 (Jan.-Feb.) 1953.

This new chart consists of paragraphs of reading material in standard gradations in size of print, one paragraph to a page. The material includes not only tests for adults, but also for children.

7-3

Smolik, H.: *Demonstration eines einfachen Apparates für das Orthoptiktraining,* Ophthalmologica, 125:419-421 (April-May) 1953.

The author describes an instrument permitting him to turn two parallel plane mirrors around their vertical and horizontal axes and as a source of light he uses Leitz' instrument for projection of small slides. The slides are placed in a way that two corresponding slides of a stereoscope, one with a red filter, the other with a green filter, are in one frame. In this way only one picture, either red or green (exactly complementary colors), is projected on each mirror and from there reflected on a screen. The separation of the two pictures (both horizontal and vertical) can be varied at will and measured on a scale. Flashing is made possible by means of a rotary disk with two semicircular openings. As it turns, the two pictures are projected successively.

The screen can be placed at various distances. Generally, it is placed at 30 cm. and 5 M. At short distances a hemispheric screen should be used. The instrument enables the study of the field of vision and scotomas and it is especially interesting in cases of squint with amblyopia.

One of the advantages of this instrument is that the parents can observe the success or failure of the children. False localization, which is very difficult to explain to a layman, can be easily demonstrated. Dr. Smolik foresees several changes and improvements in this instrument, such as the use of polaroid glasses instead of the red and green glasses.

8-1

Abraham, Samuel V.: *Special reactions to the miotic, Floropryl,* Am. J. Ophth., 36:1122-1123 (Aug.) 1953.

The author uses Floropryl and other miotics in the treatment of certain cases of convergent strabismus. He presents the cases of two children who, when treated with Floropryl 0.01 per cent, had unfavorable general body reactions.

Floropryl is effective as a miotic but in sensitive cases a sufficient amount may be absorbed through the mucous membrane of the conjunctiva or nasal area resulting in toxic effects.

Iris cysts have been noted to develop even with only 0.0005 per cent solution. The author reports that when the Floropryl treatment is stopped, the cysts disappear. Since similar smaller cysts appear in adults using pilocarpine or

eserine, it cannot be said that these cysts are produced only by Floropryl. The author's finding so far suggest that only when the pupils remain constricted do these cysts develop.

Children should be watched carefully when using miotics and treatment modified if cysts develop. When general body reactions appear, use of the drug should be stopped.

9-1

Berens, Conrad, and Zerbe, Jean: *A new pinhole test and eye-dominance tester,* Am. J. Ophth., 36:980-981 (July) 1953.

The authors describe a new paddle-like device to be used for making the pinhole visual acuity test and the eye-dominance test. The importance of both these tests is emphasized and instruction for using the tester is included.

9-2

Ruedemann, A. D.: *Foveal co-ordination,* Am. J. Ophth., 36:1220-1224 (Sept.) 1953.

Regarding the understanding of the extraocular muscles, Dr. Ruedemann says, "The entire problem is shrouded in the mystery of the fovea, its sensitivity levels, its relative superior and important recording center in the brain, and finally the development of the fusion center." In order to help understand the problem of foveal co-ordination, or more important, the lack of foveal co-ordination, the author, with the Kresge Foundation, studied the visual levels of children in high, intermediate, and grade schools.

The study revealed that the visual patterns were set by the time the child was in the first grade of school. Bad patterns became just as firmly set as good eye brain patterns. The bad pattern should be corrected as soon as possible in order to obtain correct foveal co-ordination. Personality and learning problems are often secondary to visual inco-ordination. It was found that "20 per cent of the pre-school group had some lack of foveal co-ordination."

Accommodative errors and some cases of low visual acuity can be corrected without surgery. Other cases need surgery and immediate thorough binocular re-education. "It is my contention that complete foveal co-ordination, that is constantly maintained, is the most important learning sense of the individual."

9-3

Wheeler, Maynard C.: *Strabismus; review of the literature,* A.M.A. Arch. Ophth., 50:109-122 (July) 1953.

Dr. Wheeler, in presenting a review of the literature on strabismus for the past year, pays tribute to Dr. Richard Scobee and highly recommends the second edition of his textbook "The Oculorotary Muscles."

Considerable interest in the writings of Burian and Swan is being shown in Great Britain. Both Stanworth and Boyle, following Swan's method of investigation for peripheral fusion, reached the following similar conclusions; (1) in an infant, peripheral fusion probably develops first, (2) in amblyopia, it may be peripheral fusion that holds the eyes straight, (3) it also may play a part in straightening the eyes of an accommodative squint, and (4) possibly the gradual encroachment on the macula from the periphery as a form of fusion training is suggested.

Dyer and Bierman assert that "suppression is associated with abnormal neuronal discharges in the cortex and suggest that electro-encephalographic tracings may help in the diagnosis of suppression amblyopia." Fixation anomalies in amblyopia have been investigated by Brock and Givner who found, by a new method capable of more accurate measurement of fixation, that 20 per cent of amblyopic eyes fixate along the central foveal axis. "Most fixate eccentrically, the shift away from the center being greater the more profound the amblyopia."

Anomalous Correspondence. Halldén, in a 90-page monograph on the subject of fusional phenomena in anomalous correspondence, concludes that if strabismus with harmonious anomalous retinal correspondence is operated on, lack of harmony results. The fusion will attempt to compensate for this in several ways; by a fusional movement (returning the eyes to the original position), by sensory fusion (creating a new anomalous area and lack of harmony), or by a combination of the two. Kretzschmar's study showed that the subjective angle remains essentially the same over the entire area, indicating that local signs change throughout most of the binocular field. Starkiewicz noted the importance of the position of the patient's head in taking the afterimage test. He believes that the afterimage will show signs of developing anomalous correspondence before other tests. Shepherd studied 1,104 cases to determine the prognosis in anomalous retinal correspondence and favors preoperative orthoptic training. He feels that if normal correspondence is not achieved before surgery a small residual angle may tend to return to the preoperative angle.

Methods of Examination. Capobianco suggests that testing the subjective as well as the objective nearpoint of convergence is of value in the prog-

nosis of convergence insufficiency. New methods of testing ocular deviation are reported by Kretzschmar and Falkowska. It was pointed out by Thompson that while the troposcope distance measurements are consistently higher than those of the prism cover test, the near measurements by both tests are essentially the same.

Esotropia-Exotropia. A new syndrome is described by Gittoes-Davies who also gives the possible etiology, treatment and prognosis of 80 cases. The name "concomitant strabismus with manifest diplopia" is suggested by Meunier for acute concomitant strabismus in an adult. Francheschetti, who reports 6 cases of this type, emphasized that diplopia does not always mean paralysis. A symposium on intermittent exotropia was given at the joint meeting of the American Orthoptic Council and the American Association of Orthoptic Technicians in 1951 to bring the subject of exotropia up to date.

Alternating Hypertropia. Schlossman favors Posner's explanation of alternating hypertropia as an aberration of the postural tonus and cautions against operations for the vertical muscles. The conclusions of Crone who reports 80 cases under the title "alternating hyperphoria" are of considerable interest because of his wide experience.

Nonsurgical Treatment. Abraham adds 88 cases to the previously reported 44 cases in which miotics had been used. In 93 per cent of the cases of intermittent squint and in 35 per cent of the cases of constant squinters a distinct advantage was noticed—the squint disappeared or became less frequent. He also reports the use of miotics in cases of amblyopia. The importance and success of occlusion for the treatment of amblyopia is accepted by all. Baird begins total occlusion as early as 4 months and continues until surgery is indicated or until the child is old enough for orthoptic training. He feels that the results of treatment by occlusion and preoperative orthoptics far surpass surgery and glasses alone. A new method of treating eccentric fixation presented by Fritz, has brought surprising results. Under the title "nystagmus treated by orthoptics" Healy describes 4 patients with congenital nystagmus and strabismus. Orthoptic training effected cures in these patients (one had surgery). She believes the nystagmus to have been an anomaly of the fusion mechanism. A report of 51 cases of squint is presented by Scott in which she considers the relationship between the age of onset and the age at the time of surgery as an important factor in the prognosis for single binocular vision. In a review of 40 orthoptic cases one to ten years after training, Engle concludes that the majority received benefit from the training and held on to it. Douglas has written two articles in which he critically surveys the orthoptist. His conclusion is that orthoptics is better for diagnosis than for treatment.

Surgical Treatment. Dr. Wheeler found surprisingly few articles on the surgical aspect of strabismus but makes a comprehensive report on several which appeared in 1952.

A brief report is made of the symposium on the treatment of concomitant esotropia at the 1952 Oxford Ophthalmological Congress.

10-1

Anderson, J. Ringland, *Causes and treatment of congenital nystagmus,* Brit. J. Ophth., 37:267-281 (May) 1953.

The author has divided congenital nystagmus, according to the type of movements, into three groups, (1) the visual or fixation type in which there is an oscillating movement, (2) oculomotor, which is rhythmic, with the fast component to the side of the gaze, and (3) vestibular, which is rhythmic with the fast component determined by the side, rather than the gaze. In choosing the patients in this series the author has excluded any cases which might have been caused by maternal measles.

Among the characteristics of congenital nystagmus are usually poor vision, oscillating movements in the position of rest, and rhythmic movements on conjugate deviation, occasional rotations of the 12 o'clock position of the limbus either clockwise or counterclockwise, displaced position of rest, and with the former, a typical head position attempting to center the position of rest.

During the past thirty years, of 30,000 patients seen 138 of them have had nystagmus, in 106 the condition was congenital. In this period there have been two increases, the first from 1921 to 1933, the second from 1937 to 1943. It is supposed that the first increase was caused by the advent of twilight sleep. The second increase may well have its origin in the war.

Out of the 87 cases in the series 13 were considered inherited. The other causes were maternal ill health and labor difficulty, prematurity, natal anoxemia and injury. The most important of these were natal anoxemia and injury, particularly anoxemia. The main findings are presented in a tabular form giving birth information, maternal health, etc.

In treating congenital nystagmus it must be pointed out to the parents that these children need a lot of rest and freedom from undue strain. The treatment should be sightsaving classes for those with poor vision, orthoptic

treatment when indicated, full correction or overcorrection with bifocals, and surgical treatment of strabismus.

The author concludes by pointing out that a spontaneous cure often results with the passage of time.

11-1

Cushman, Beulah, and Culver, James: *Prolonged occlusion,* Am. J. Ophth., 36:76-81 (Jan.) 1953.

The prolonged occlusion test was introduced by Marlow in 1924 and is known as the Marlow occlusion to distinguish it from the occlusion of the good eye in the treatment of amblyopia ex anopsia. Marlow explained its use as being the screen test extended in point of time. It should be carried out until the position of rest is achieved, i.e., the measurements are the same from day to day, but as a rule a week's occlusion will furnish valuable information.

The authors used prolonged occlusion routinely on all patients whose asthenopic symptoms could not satisfactorily be relieved by the routine tests. The patch was left on for a period of from forty-eight hours to a week, and it was emphasized that the occlusion had to be total, taking care to carry out the dissociation even during diagnostic procedures. There was often relief of the asthenopic symptoms after forty-eight hours of occlusion. At no time has the prolonged occlusion ever brought out any muscle anomaly in patients with a normal muscle balance. A patient with a normal muscle balance can be occluded for weeks and no strabismus will become evident.

Fifty-one patients were treated with prolonged occlusion over a period of two years, and only two were found to have no muscle anomaly; twenty-four were treated with surgery, and 10 with prisms.

12-1

Adler, Francis Heed: *Pathologic physiology of strabismus,* A.M.A. Arch. Ophth., 50:19-29 (July) 1953.

The author has made an analysis of how pathologic processes affect ocular motility. The neuromuscular mechanisms which control eye movements are supranuclear, nuclear, and infranuclear. Patients with strabismus are usually divided into two groups, comitant and incomitant. If incomitant, the pathologic process causing it is situated in the nuclear and infranuclear portion of the pathway. Comitant strabismus, always innervational in origin, is caused by a disturbance in the mechanism dealing with vergences; it is therefore a supranuclear lesion.

Experiments carried out by Dr. Yasuna, Dr. Nulsen, and the author with the surgical help of Dr. Scheie and Dr. Ojers prove that lateral rectus muscles are actively innervated during divergence. Insulated bipolar electrodes were inserted into one of their lateral rectus muscles. Changes in electrical potential and the number of impulses discharged per second in various positions of gaze were recorded. These experiments prove that divergence is due to cocontraction of the lateral rectus muscles with simultaneous inhibition of the medial rectus muscles. "A divergence mechanism, therefore, exists."

Two groups of tonic impulses keep eyes in alignment; postural reflexes and optomotor reflexes. Loss of vision in childhood or paresis of vertical muscles sufficient to dissociate the eyes so that fusion is not possible may cause esotropia due to excessive tonic convergence tonus. Alcohol and anoxia cause an increase in convergence tonus for distance; therefore, it is possible that the convergence mechanism is kept under cortical inhibitions. Other causes of strabismus are due to trauma in birth, cerebral palsy, lack of myelination, etc.

Excessive divergence impulses is a probable explanation of cases of intermittent exotropia where neither refractive error nor sensory problems are the cause. Interference with the normal divergence innervation could account for esotropia in some infants who appear to have no abduction but who can with each eye abduct normally. The excessive tonic convergence may cause esotropia because of the dissociation created by weakness of divergence.

13-1

Hulsman, Helen L.: *Visual factors in reading with implications for teaching,* Am. J. Ophth., 36:1577-1586 (Nov.) 1953.

The school screening tests for reading difficulties are not adequate. Two tests considered by the author to be efficient are the Massachusetts Vision Test and the Eames Eye Test, both containing the Snellen component. However, the Snellen chart locates only 60 per cent of the children needing visual help because only distance vision is tested.

Several of the common eye defects are discussed including refractive errors and phorias. There has been a difference of opinion among those in research as to the relationship of eye defects and reading difficulties. Visual defects

among poor readers have been spotted in several studies. One prominent fact in the Eames study was "the poor reading and ophthalmic group presented medians of six prism diopters among the unselected cases." Robinson supports this finding and agrees with others that hypermetropia also has a high incidence among poor readers. However, Traxler states that "the relationship between visual defects and reading disability is not as high as one would naturally expect it to be." Robinson found aniseikonia to have a high incidence among poor readers but along with others tends to minimize the importance of myopia as a deterrent to good reading. Other studies find amblyopia, but not astigmatism, to be important in reading disabilities. The author reports on several more studies which have investigated other possibilities for reading troubles.

In view of the studies reported, the author feels that the visual acuity of a child with reading difficulties should be tested by an oculist. If correction of the defective vision does not solve the problem, at least the child will be subjected to less visual strain.

The author suggested that eye tests be given after a play period rather than after a study period. A discussion of the effectiveness of glasses follows. Several suggestions are presented for teachers in order to aid their pupils.

14-1

Asher, H.: *Suppression theory of binocular vision,* Brit. J. Ophth., 37:37-49 (Jan.) 1953.

The purpose of the twelve experiments contained in this paper is to reconsider, and reinforce, du Tour's opinion in relation to depth perception; that is, that there is always suppression of one of a pair of corresponding points, and not actual fusion. There are twelve diagrams shown and a discussion follows each.

The author states that there are two kinds of suppression: namely, suppression of ground and suppression of object. These separate suppressions are necessary to avoid the occurrence of intermediate hues and of diplopia, respectively. Following du Tour's theory it would mean that binocular mixtures of colors could not occur. However, the views that these mixtures can be produced has been brought out by others and these views are carefully examined.

The following are the author's statements concerning the suppression theory of binocular vision.

The correspondence of the retinal elements is comparatively rigid and unchanging.

One of a pair of corresponding points always suppresses the other.

Where there is a contour, the suppressing power of retinal elements on each side of it is enhanced.

Where there is disparity of contour, there in the one eye retinal elements on both sides of this contour suppress corresponding points in the other eye.

If the extent of the suppression is greater than the disparity between the contours, then diplopia occurs, but depth perception is possible.

If the extent of the suppression is greater than the disparity between the contours, one contour is suppressed and single vision occurs with depth perception.

The contour of one part of the image may be dominant in one eye, and that of another part may be dominant in the other eye.

15-1

Leurent, Par M.: *De l'intérêt de diverses méthodes d'examen dans l'étude des paralysies oculaires; le chéiroscope comme élément d'appoint au diagnostic,* Bull. Soc. d'opht. de France, 2:67-81 (Feb.) 1953.

The author suggests the use of the cheiroscope for the diagnosis of complicated cases of paralysis. He gives an example of a case with a paralysis of the right superior oblique and describes the different tests he used for the diagnosis such as the Maddox rod, the synoptoscope, the Hess screen and the cheiroscope.

The patient traces a picture on the cheiroscope and then the author traces the same picture on the same sheet of paper and compares the results. If the figure drawn by the patient is displaced to the left, higher and tilted to the right, and the vertical displacement increases on looking left, the author concludes a paralysis of the right superior oblique.

The cheiroscope gives results easily and quickly. It helps in the diagnosis of cyclophoria and this makes possible the immediate differentiation between an oblique and a rectus muscle.

The patients usually like this test, although sometimes macular suppression makes it more difficult.

16-1

Giardini, A.: *Sulla terapia ortottica e chirurgica dello strabismo concomitante.* Boll. d'ocul., 31:396-421 (July) 1952.

The article is a didactic one about concomitant strabismus and is built up according to the data reported in the recent literature and experience of the author. The following resume concerns only the orthoptic part.

It is the belief that orthoptic treatment and

occlusion should be done as early as possible to be effective. Orthoptic treatment should be prescribed preoperatively and postoperatively. Preoperatively, its aim is to eliminate improper fixation, suppression, anomalous correspondence (especially in the cases of so-called deficiency of retinal normal correspondence), to improve the power of fusion and of convergence, etc. Postoperatively, orthoptics is used to correct unavoidable residual postoperative deviations by reinforcing the power of fusion.

In nonaccommodative esotropia the author states that he saw few cases of true anomalous correspondence but usually deficiency of retinal correspondence, respectively normal or anomalous (either harmonious or unharmonious). The first condition is obtained when the squint is able to obtain superimposition of the image at the objective angle (normal) or at different angle (anomalous). The second condition exists when there is crossing of the images instead of superimposition.

Amplitudes of convergence and divergence should be given to the esotropia and exotropia. It is an error to give only divergence to the esotropia and only convergence to the exotropia. Fixation and diplopia exercises are useful exercises for patients who have the ability to fuse.

The accommodative esotropia is given a full correction, and bifocals are prescribed in certain cases. Partial occlusion of the inferior half of the lens corresponding to the better eye may be used. Dissociation of accommodation and convergence is prescribed following antisuppression exercises. If the refractive error is less than 3D and the astigmatism is not bigger than ID it is possible to teach the patient to keep his eyes straight while accommodating without correction.

In the intermittent exotropia, it is advisable to modify the static position of the eyes with surgery adding preoperative and postoperative orthoptic training. In the exotropia with so-called excessive divergence aim of the orthoptic treatment is to obtain voluntary control of the eyes. To have a favorable definitive result this effort of control has to be helped with surgery. Occlusion is seldom necessary. The macular suppression is frequently very resistant to therapy. The sequence of treatment is often inverted since the author found that it is easier to fuse similar images rather than superimposition of dissimilar images. The size of the targets is gradually decreased.

In the constant exotropia, being usually adult people with fusion, there is indication only for cosmetic surgical correction.

16-2

Kent, Paul R., and Steeve, J. Howard,: *Convergence insufficiency: incidence among military personnel and relief by orthoptic methods,* The Military Surgeon, 112:202-205 (Jan.) 1953.

A series of 4,461 patients at United States Naval Hospitals in San Diego, Calif., and Camp Lejeune, N. C., were examined to determine the incidence of symptoms producing exophoria and to form an opinion as to the practicality of orthoptic measures.

In addition to either a cycloplegic or manifest refraction, each patient had his vertical and lateral phoria measured at 20 feet and 13 inches with a Maddox rod and Risley prism. Prism convergence at 13 inches with break and recovery point and the N.P.C. to the bridge of the nose were also recorded. Those chosen for orthoptics had movements in eight positions, cover tests for 20 feet and 13 inches, and interpupillary distance measured and recorded. Those whose phorias were of structural rather than innervational origin were not treated orthoptically.

The criteria by which patients were chosen for orthoptics were ocular asthenopia unrelated to refractive error or pathology, and the presence of one or more of the following: prism convergence at 13 inches less than three times the amount of exophoria for the same distance, prism convergence at 13 inches of less than 15 prism diopters regardless of phoria, or a prism recovery point at 13 inches of 5 prism diopters or less.

The main symptoms of which the patients complained were headaches, blurring of vision, ocular fatigue, occasional diplopia, nausea, burning, epiphora, sleepiness and loss of concentration. All of them had low prism convergence at near.

Orthoptic training was carried on for twenty minutes once a week. Prisms of increasing strength were held first in front of the right eye, then in front of the left eye, using a light or target at 20 feet for fixation. Those who had a remote N.P.C. were also taught to use the "pencil-pushup" exercise. The results began to be noticed after about three weeks of training, and within about eight weeks the results were almost uniformly good. Those who have been re-evaluated since training have retained the convergence which they built.

16-3

McGannon, William J.: *Teletrainer,* A.M.A. Arch. Ophth., 50:354-355 (Sept.) 1953.

Television may be used as an antisuppression home training method to maintain the interest of the patient. A polaroid screen is placed in front of the television screen, one half at an axis of 90 degrees and the other half at an axis of 180 degrees. The patient wears polaroid lenses matching the two sides of the screen. Simultaneous perception is necessary for the entire picture to be seen.

16-4

Parfornt, Jean and Polliot, Louis: *Le traitement chirurgical du strabisme concomitant.* Societe d'Ophtalmologie de Paris, Rapport Annuel, Supplement au Bulletin d'Ophthalmologie de Paris. No. 4, 1953.

The advantages and disadvantages of early and late surgery are discussed. The reasons listed in favor of postponing the surgical procedure until after the age of puberty are (1) a spontaneous straightening of the ocular axes occurs quite often, sometimes several years after the correction was prescribed, (2) secondary deviations are more frequent in patients operated upon at an early age, (3) in spite of the great progress in the administering of anesthesia, it is still dangerous for young children and (4) under anesthesia or even after retrobulbar injection the deviation is much smaller, and sometimes disappears completely. Therefore, under general anesthesia it is impossible to control the correction adequately. Many surgeons give rules for the number of millimeters to resect or recess for a certain deviation but these rules are very unreliable and vary from surgeon to surgeon. Those in favor of an early intervention claim that (1) the correction of the deviation is more stable because of binocular vision, (2) it prevents secondary changes, muscular and sensory (amblyopia ex anopsia, and anomalous correspondence) and (3) it prevents the development of an inferiority complex in the child.

In an answer to those in favor of early surgery the authors state: Binocular vision per se is not important enough to justify an early intervention. Although much is written and said about the sensory changes following strabismus, the authors are not convinced that they really exist.

Regarding amblyopia, two types of amblyopia are discussed, namely, true and relative. True amblyopia is found in patients with less than 20/400 visual acuity and absence of central vision. This type of amblyopia cannot be improved.

Relative amblyopia refers to visual acuity of 20/200 or better and it may be improved either by training or spontaneously but will not disappear completely.

Some children are very much disturbed by occlusion and will not wear it under any circumstance. They are usually blamed for the failure if there is no improvement in visual acuity, but in reality it cannot be improved. On the other hand, when a child does not mind wearing an occluder and does not peek and there has been some improvement in visual acuity it means that his visual acuity was much better than the doctor was able to bring out on the first visit.

The authors believe that retinal correspondence is innate and cannot be modified. Even if a new anomalous correspondence could develop, it would need a constant, never-changing angle of deviation while actually the angle between the two visual axes changes constantly when the patient looks from a near to a distant object. What really occurs is strong suppression in several zones that can be detected by studying the binocular visual field.

An early surgical intervention would not make much difference anyway because the visual axes have to be adjusted with microscopic accuracy to ensure good correspondence and this is not possible with the crude surgical technics.

The psychologic factor does not seem to have very much importance. Psychologic tests were conducted with strabismic children that came to Troussean Hospital for one year. These tests showed the children had normal intelligence and social behavior. Of the 1,000 children that were examined for psychiatric consultation the percentage of children with strabismus was only 2.2 per cent which was exactly the normal proportion. As for orthoptics its result does not justify the continuous effort of both the parents and the children. On the other hand, in specialized centers when it is possible to do orthoptics without interrupting the other activities of the child, its theoretical possibilities are very interesting. Orthoptic studies and methods can help to understand the problems of binocular vision.

17-1

Ogle, Kenneth N., and Prangen, Avery de H.: *Observations on vertical divergences and hyperphorias,* A.M.A. Arch Ophth., 49:313-334 (March) 1953.

Normal vertical fusional amplitude is 2.5 to 3, yet it can be shown that people are able to adapt to larger degrees of vertical divergence.

The fixation disparity technic was given in detail. Hyperphorias were forced into vertical divergence by overcoming prisms which they

compensated for completely, most exceeding 6 in steps of a 2 gradual increase. No changes were seen in the original hyperphoria when complete vertical divergence had been obtained. After prism fit-overs were removed, diplopia was evident from two to sixteen minutes after the recovery of fusion. (This varied with the individual subjects.) When the stimulus for fusion was delayed, the hyperphoria did not return to normal even after two and a half hours. Compensation among hyperphorias varied, some accepted the prisms in exactly the degree prescribed and maintained their hyperphoria as originally measured.

We constantly use vertical divergence in everyday life, as in near vision, when reading and then looking obliquely at a fixation point on the page of a book without turning our head and seem to compensate for it automatically.

The correction of an anisometropic refractive error is readily accepted by many patients, when changing fixation from distance to reading position with the same hyperphoria in all directions of gaze constantly demanded.

Normal binocular impulses seem strong enough to overcome artificial obstacles such as prismatic deviations, anisometropic refractive corrections, or vertical prisms with no change in the original hyperphoria. The psycho-optical and the voluntary reflex seem to stimulate the compulsion for the fusion reflex to act. The fact the fusion reflex is strong enough to compensate for large vertical prisms, yet is unable to overcome the hyperphoria, can only be explained as an obstacle to motor or innervational abnormality of the ocular muscles. This does not take hyperphorias of paretic origin into consideration.

18-1

Ogle, Kenneth N.: *On the problem of an international nomenclature for designating visual acuity,* Am. J. Ophth., 36:909-921 (July) 1953.

This paper is a report of a subcommittee of the American Committee of Optics and Visual Physiology on the problem of devising a system of designating visual acuity. The committee was asked "to work out the geometrical and decimal systems together in a practical manner." The committee studied the problem of finding and standardizing "(1) a particular mode of designating visual acuity and (2) a particular gradation of steps in letter size for the visual acuity charts." Detailed tables, graphs and computations are included.

18-2

Stern, Richard U.: *Method of testing visual acuity in children,* Tr. Am. Acad. Ophth. 57:686 (Sept.-Oct.) 1953.

Dr. Stern is of the opinion that most charts for testing children's visual acuity are unreliable because of the variability of responses and children's short attention span. He feels that the use of the isolated "E" with the child holding an "E" is very satisfactory and fairly free from the faults mentioned. The test is easily explained and holds the child's attention. He feels that this test gives a valid measurement of visual acuity even in very young children.

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